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XXIV.

**PATHOLOGY AND INTRAMURAL ELECTROCOAGU-
LATION OF THE INFERIOR TURBINATE.**

BY JOSEPH C. BECK, M. D.,

CHICAGO.

The discussions about the inferior turbinate are very infrequent in our societies compared to other intranasal structures, because most everything about it is fairly well settled, especially since the conservatism in its management is agreed upon. To one who has been in the practice when the inferior turbinate was blamed for many ills, and its treatment was so varied, including the wholesale removal, it is a great satisfaction to look back and reminisce a bit.

When the late Dr. Dudley Reynolds of Louisville, Ky., presented at one of our national societies a collection of inferior turbinates (400) that he removed for the cure of all sorts of diseases, both local and general, the discussion that followed was so definitely adverse to his conclusions that it was, I believe, the death knell to the indiscriminate surgery of this vitally important physiologic structure. On the other hand, the enlargement of the inferior turbinate, when constant and due to chronic and permanent pathologic changes, is capable of

producing symptoms that are very annoying to the patient and require correction.

Since the conservative method of treatment has become established, I feel that its interpretation has gone too far and now too much tamponing, smearing and spraying is being done with the expectation of permanent relief of symptoms. It is with this point in mind that I wish to review the pathology and present a simple type of conservative treatment, developed by my associate, Dr. M. R. Guttman, which I hope will be more satisfactory than any we have employed heretofore.

Permit me to refer to some of the semisurgical methods that have been and still are in vogue:

- (a) Galvanic cautery in all its modifications.
- (b) Submucous section of blood vessels and pressure.
- (c) The use of the angiotribe on the lower and posterior portions.
- (d) Intramural deposits of chromic acid.
- (e) Partial removal of the lower border and the amputation of the posterior ends.
- (f) Attempts are made to remove the excess, including some of the bone, and then to suture to avoid granulations and prolonged healing.

There are still too many partial removals of the anterior ends of the inferior turbinate in connection with antrum operations.

Enlargements of the inferior turbinate, due to acute inflammations, are not discussed, since they are not within the province of this paper.

PATHOLOGY.

Grossly, it is not easy to determine the exact pathologic state of the inferior turbinate simply from its enlargement, and many times there is a combination of changes in the one structure. However, we can classify them into:

1. Vascular type, known as turgescence, easily contracted by cocain, adrenalin and ephedrin.
2. Hypertrophy of both the bone and soft parts, which is not easily contracted by cocain, adrenalin or ephedrin.
3. The pale, smooth and noncontractile body, so called hyperplastic or allergic form, usually associated with a similar pathology of the middle turbinate and sinuses.

4. Atrophic inferior turbinate.

5. Neoplastic and other rarer forms of inflammation, as tuberculosis, lues and rhinoscleroma.

The only way one can determine the exact changes is microscopically. For practical purposes, however, one need not have a biopsy in order to treat such conditions. It is mainly necessary to know the minutiae in order to appreciate what result one may logically expect from treatment in such conditions.

INTRAMURAL ELECTROCOAGULATION.

The object that is desired to be obtained by intramural coagulation of the turbinates is to secure a decrease in the volume of the soft tissues by the incorporation in them of a shrinking scar. In addition, this scar when produced prevents turgescence in event the turbinal becomes engorged. The principle is not new. Linear cauterization of the turbinals with the electric cautery is familiar to all. This procedure (electric cautery) is attended by several drawbacks. It may be accompanied by a marked reaction that may last for a long period of time. The resulting nasal obstruction and discomfiture is resented by many patients. Scab formation and severe secondary hemorrhage may occur. Respiratory epithelium is destroyed and in some instances synechia may form between the septum and turbinates. None of these drawbacks have been noted with the method advocated. The reaction following this procedure is surprisingly mild and accompanied by little or no discomfiture. Since the epithelium is not destroyed no scab or synechia formation is noted. Since the coagulating current is used the vascular beds are sealed and hemorrhage does not occur. Pierce's method of intramural cautery with chromic acid is not always followed by positive results.

TECHNIC.

The instrumentarium is simple. Any one of the standard diathermy machines that is sufficiently heavy for surgical coagulation is used as the source of the current. At this time it might be well to state that a number of very small apparatuses have been offered to dentists and otolaryngologists that have proven to be failures in that they do not produce a heavy

enough current. In addition one requires a large indifferent electrode and a special turbinate needle electrode.

This turbinal electrode, as designed by my associate, consists of a long Hagedorn needle that has been covered, except for about two millimeters at the point and by a special dielectric compound. This needle fits any of the standard electrode holders such as the Plank holder. The machine is adjusted so as to produce coagulation current, the strength of which is enough to blanch an area of one or two millimeters in diameter. It is obligatory, in our practice at least, to test out the current on a piece of meat. When a proper adjustment is obtained the indifferent electrode is connected to the patient's back or arm. A good contact must be assured. In the meantime anesthesia of the turbinate is effected by a 10 or 20 per cent solution of cocain. There is one objection to the use of cocain in that it produces a marked shrinkage in the intumescent types of turbinates that may make the introduction of the needle difficult.

Recently we have been using nupercane, a quinin derivative, that does not cause shrinkage but effects a good anesthesia. The anesthesia being satisfactory, the special needle is plunged into the head of the turbinal in about the center and carried along the medial aspect to the posterior part of the inferior turbinated body, hugging the periosteum.

The current is now turned on with a foot switch and the needle slowly withdrawn, the current acting all the while. Just before the needle point is withdrawn the current is broken. This causes a line of coagulation about two millimeters in diameter to be formed in the cavernous tissue the entire length of the turbinate which, after healing and replacement by a shrinking scar tissue, will effect a reduction of the medial aspect of the turbinal body away from the septum. The same procedure is carried out along the inferior aspect of the turbinate, which will cause the tissue to shrink up away from the floor of the nose after contracture has occurred. In this work the spark gap adjustment must be very smooth, as an irregular, jerking gap action will produce unpleasant and painful galvanic effects that may cause the patient to jerk his head about.

Usually the two lines of coagulation described will suffice, but in large turbinals one may place two lines of coagulation

along the medial aspect of the turbinate and two along its inferior portion, as best suits the judgment of the operator. The postoperative care is merely that of observation. Within two to four weeks following the procedure one will notice the indented appearance of the turbinals. The patient will experience relief much earlier than that. This is especially true when the pathology is that of the intumescent type of alternating nasal obstruction.

REPORTS OF CASES IN GENERAL AND FINAL RESULTS.

Over fifty cases of bilateral intumescent and hypertrophic inferior turbinates have been subjected to this procedure since August, 1929, and while sufficient time has not elapsed to judge as to the permanency of results, one may make a few pertinent observations.

As a consequence, healing with dimpling of the turbinate, due to the formation of submucous scar, appeared within four to six weeks. Relief from nasal obstruction was secured in practically every case. Few cases needed a subsequent coagulation. This was necessitated in very large turbinates, some of them being almost the thickness of one's thumb. A few cases in which no relief from obstruction was obtained necessitated a subsequent septum resection, or as in one case, the removal of a persistent adenoid mass in an adult.

A detailed statistical review of the results obtained, together with histologic studies of the effects of the intramural coagulation, will appear in a subsequent contribution in this publication.

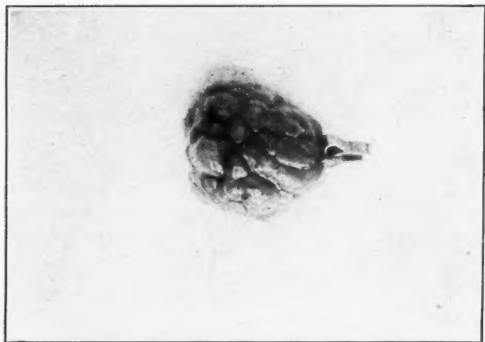


Fig. 1. Mulberry hypertrophy of the inferior turbinate (post. end) showing undulation due to scar formation and contraction. In reality the projecting masses are atrophic as to functional tissues as glands and erectile tissue is concerned.

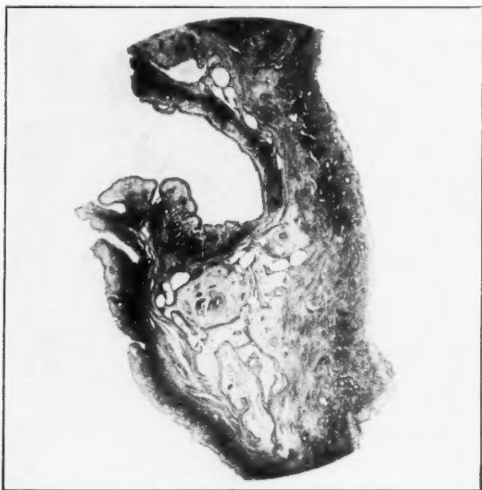


Fig. 2. Hypertrophy of the inferior turbinate showing the irregularities and degeneration of the mucous membrane. The bony spacing appears to be increased from the type considered normal.



Fig. 3. The same as Fig. 2—Higher power showing the massive connective tissue formation. Very thick degenerated epithelial covering and great paucity of glandular elements.



Fig. 4. So called hyperplastic (waterlogged) inferior turbinate showing marked edema with distended lymph channels and cystic dilations. The epithelial covering appears in a state of cloudy swelling—non-differentiating.



Fig. 5. Same as Fig. 4. Higher power—showing particularly the marked lymphatic infiltration in regular channels. The poor staining of the edematous tissue.

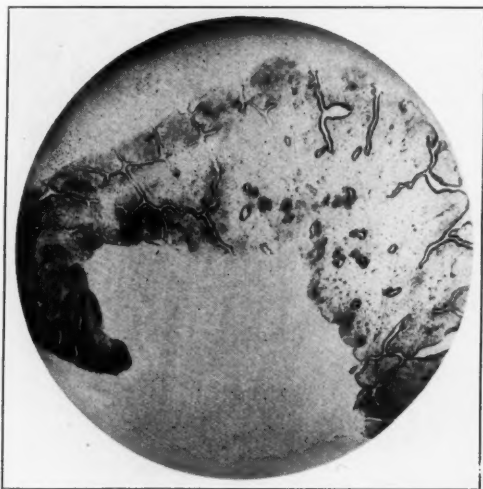


Fig. 6. Marked hypertrophy with a great deal of folding in of the epithelium, giving the appearance on section of channels. These are pure artefacts due to folding and overcrowding within the nose.

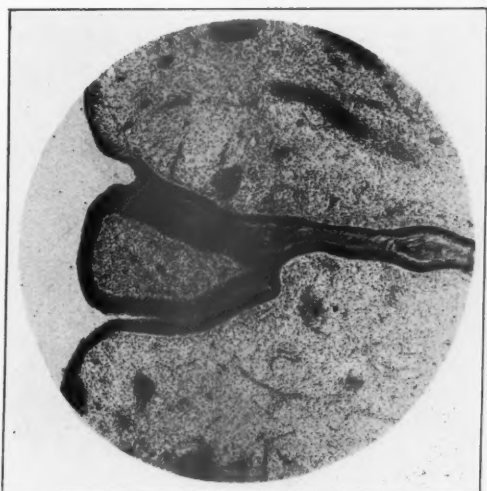


Fig. 7. A section of the same as Fig. 6. Higher power—showing one of those channels of epithelial folding—here is shown actual hornification, degeneration and edema of the chronic type.

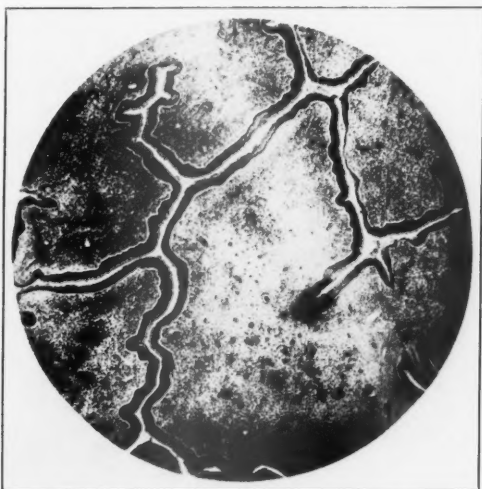


Fig. 8. Same as Fig. 6. Higher power—showing very grotesque channel formation of the folding in of the tissues. The epithelium is very thick—stains well, yet does not show differentiation.



Fig. 9. Hypertrophic inferior turbinate. The bone appears greatly spaced. These marrow spaces appear cystic. The soft parts are mostly chronic fibrosis and very few glands are present.



Fig. 10. Same as Fig. 9, showing only the bony section. Large blood vessels within the bony spaces, which show great vacuolization, probably artefacts. The bone is much thinned out by this increase in marrow spaces.



Fig. 11. Typical section in an inferior turbinate in atrophy. The epithelium and subepithelial tissues are very thin on the septal side. The bone is much atrophied. On the antral side tissues, especially, the glands are preserved.

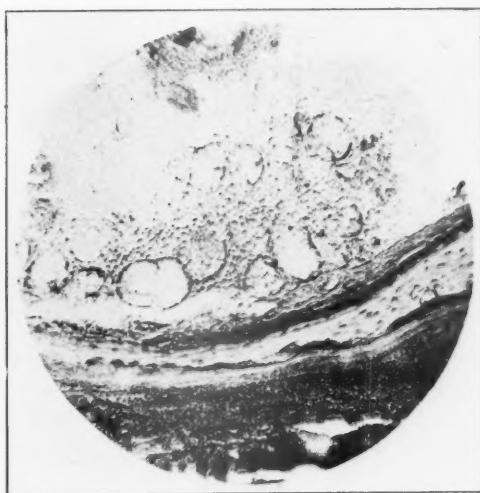


Fig. 12. Same as Fig. 11 with higher power. The metaplasia of the epithelium is well demonstrated. The bone is thin but not much inflamed. The glands are in a state of degeneration.

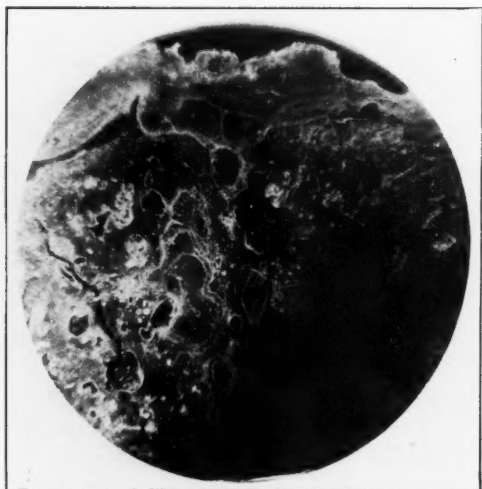


Fig. 13. Section from the inferior turbinate that had been treated by the angiotribe (crushing) two weeks after, showing marked reaction not recognizable as to cellular details.



Fig. 14. Section from the portion of the inferior turbinate that had been treated by electro-coagulation by the intramural route. Note the slight reaction two weeks after.

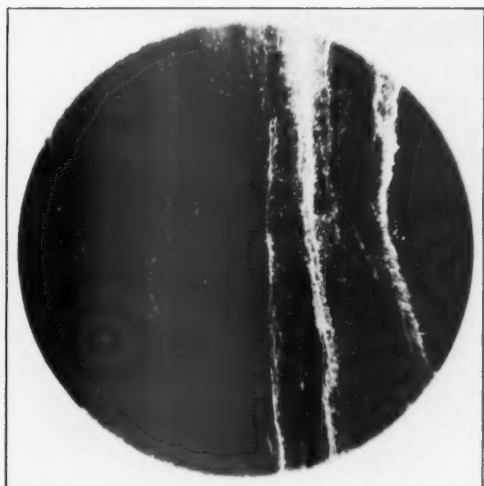


Fig. 15 Persistent exudate of the inferior turbinate following actual cautery—two weeks after—showing no differentiation of any cellular elements.



Fig. 16. Section from the inferior turbinate that had been cauterized by the actual galvano cautery—two weeks after—showing marked reaction and piling up of tissues not differentiated in cellular elements.

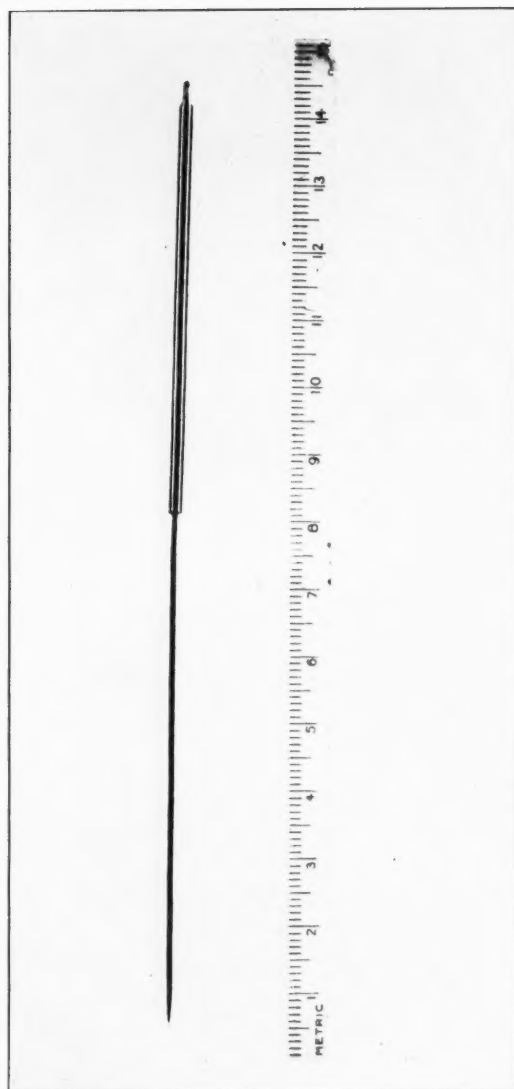


Fig. 17. The needle for intradural coagulation.

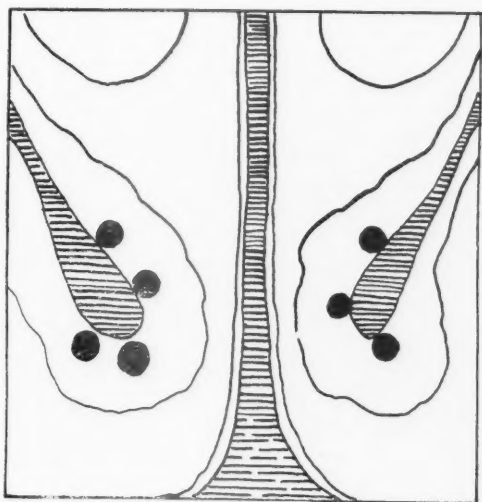


Fig. 18. Cross section of inferior turbinates showing the location of the several streaks of electrocoagulation in relation to the periosteum of the turbinate.

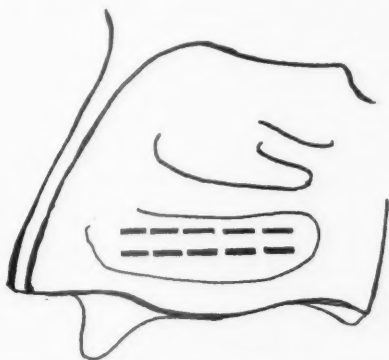


Fig. 19. Showing course of the turbinal electrode within the body of the inferior turbinate.

CONGENITAL ANOMALIES OF THE ESOPHAGUS WITH A REPORT OF NINE CASES.*

By KENNETH A. PHELPS, M. D.,

MINNEAPOLIS.

Congenital malformations of the esophagus are not exceedingly rare. Since 1696, when Thomas reported the first case, the literature contains many references to the subject. At various times different authors have collected the cases appearing in the literature. Mackenzie, in 1884, found sixty-three cases, Kreuter¹ in 1905 found 111 cases, Plass² in 1919 had 204 cases, Reynolds and Morrison³ found one case in sixteen years at Bellevue, Vinson⁴ reports 186 cases of stricture of which seven were congenital, Stukowsky and Boran⁵ found one case in an experience of 50,000. I have been able to locate many more in the literature, and I feel sure there must be hundreds of cases that are not reported at all.

Different authors have classified the varieties of congenital abnormalities of the esophagus, but I have not found a thoroughly complete classification yet published, therefore, I present the following:

- A. Absence of the entire esophagus.
- B. Esophagus represented by a solid cord throughout.
- C. Double esophagus.
 - 1. Complete.
 - 2. Partial—bifurcations joining near lower end.
- D. Spasm of esophagus.
- E. Diverticula $\left\{ \begin{array}{l} \text{Traction} \\ \text{Pulsion} \end{array} \right.$
- F. Esophagotracheal fistula (malformation of esophagotracheal septum).

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Atlantic City, May, 1930.

1. Esophagus and trachea normal, except for fistula.
2. Esophagus in two segments.
 - (A) Upper segment.
 1. Orifice normal.
 2. Lower end atresia.
 - (a) Connected to stomach or lower esophageal segment by solid cord.
 - (b) No connection to stomach or lower esophagus.
 - (c) Fistula into trachea.
 - (B) Lower segment.
 1. Upper end has fistula into trachea or bronchus.
 2. Lower end opens normally into stomach.

G. Cysts.

H. Atresia.

1. Fold or valve in mucous membrane.
2. Web or diaphragm.
3. Solid cord between segments of esophagus.

I. Stenosis.

1. Narrowing of lumen, upper, middle, lower or multiple.
2. Paralysis.
3. Pressure from without

{	lung tips
	heart or blood vessels
	thymus

Certain of these anomalies are best explained on an embryologic or evolutionary basis; others have no good explanation.

The archenteron, or primitive digestive tract, is a tube closed at each end, which is constricted off the dorsal portion of the yolk sac. As the embryo infolds at its extremities, the archenteron becomes differentiated into three parts, the fore, middle and hind gut. From the fore gut are developed the pharynx, esophagus and stomach as well as the larynx, trachea, bronchi and lungs. The upper portion of the esophagus develops from the ectoderm (Losee¹⁰) and contains striated muscle fibers; (Kieth¹¹) the lower part comes from the entoderm of the primitive archenteron and has nonstriated muscle fibers. Until the neck is differentiated, the esophagus is a mere sphincter

between the pharynx and stomach. In animals without a neck, as amphibians, the lungs spring directly from the larynx and the trachea, and the bronchi do not develop. In the human, the appearance of a neck requires a rapid elongation of the esophagus and trachea, which up to the third week form a single structure. This common origin can be seen in their common nerve supply, the recurrent laryngeal nerve.

The lumen of the esophagus in a four millimeter embryo is flattened from side to side. It is lined in the lower portion with two layers of cells. The nuclei in the cells nearest the lumen show mitotic figures. The lumen of the upper portion has a single row of cells lining the dorsal area, while the ventral has three and four layers. This thickened area is the earliest sign of the respiratory tract.

During the third or fourth week there appears a groove in the floor of the primitive fore gut. The margins of this groove fuse and form the esophagotracheal septum. The last portion to fuse is at the level of the bifurcation of the trachea. This union takes place at the same time the fore gut is being elongated. The lung buds arise from the distal end of the trachea and by their rapid development push the stomach backwards into the body cavity. This new position of the stomach necessitates the elongation of the esophagus.

After the separation of the esophagus and the trachea by the formation of the esophagotracheal septum, the esophageal lumen becomes greatly narrowed by proliferation of the epithelial lining. The esophageal lumen of a 7.5 millimeter embryo is one-half the size of a four millimeter embryo, while its length is three times as great. Formerly, some authorities believed the lumen became entirely obliterated (Kreuter¹), but since Johnson's¹² work, in 1910, it has been generally accepted that the lumen is never entirely closed. Atresia, therefore, is abnormal at any stage. In certain vertebrates, as the turtle (Reese¹³), a temporary atresia of the esophagus is a normal developmental condition. In this animal the esophagus is open first, then closed and finally reopens. In fact, the trachea is often closed also.

In twenty millimeter embryos vacuoles appear in the epithelium (Keibel and Mall¹⁴). These unite and result in a larger

lumen. At certain stages a cross section of the esophagus may appear to have several lumens, due to the size of these vacuoles.

When the embryo is ten millimeters long, folds appear in the mucous membrane which make the lumen irregular in shape. These folds twist as they go down the esophagus, so that the dorsal and ventral folds in the middle esophagus become right and left lower down. In the lower esophagus these folds are arranged in four primary ones and form a cruciform lumen.

In fifty-five millimeter embryos there are cilia on certain areas of the epithelium; however, no cilia are found at birth.

A. Absence of the entire esophagus occurs only in monsters and is not clinically important. There are eight cases reported by Cautley¹⁵ in 1917. Marsh¹⁶, in 1902, reports a case of complete absence of the esophagus. The stomach had attached to it an esophageal termination of normal caliber. Neff⁶ reports a case of absence of the middle third, the two extremities being present.

B. The esophagus represented by a solid cord is also very rare. Parrish¹⁷ reports such a case. This condition must be allied to that found in turtles, as mentioned above.

C. Double Esophagus.—Two cases are recorded by Cautley¹⁵ in 1917. No satisfactory explanation of this anomaly has been brought forth.

D. Spasm of the esophagus of congenital origin is a fairly frequent condition. It may occur in any portion of the esophagus but more frequently at its lower extremity. Many such spasms produce no symptoms, but their occurrence can be demonstrated by fluoroscopy soon after birth, as recently done by Birnberg.¹⁵ H. J. Moersch¹⁸ reports cardiospasm in children and found two cases of congenital origin.

A large number of names, as idiopathic, primary, essential, diffuse, neuropathic dilatation, functional hiatal stenosis, mega esophagus, preventriculosis, esophagectasia, phrenospasm and achalasia, have been given this condition, but as "cardiospasm" is the most common it will be used in this paper. The finding of a dilated esophagus above the spasm is almost constant. This has led to many theories being advanced. Bard of Strassburg believes the dilatation is due to a con-

genital enlargement of the esophagus, similar to mega colon or a dilated ureter. Mackenzie and Roseheim maintain that the esophageal muscles are congenitally atrophic, and the dilatation results therefrom, leaving the cardiac narrowing as a secondary phenomenon. Most authors believe the primary factor is the spastic narrowing of the esophagus and the dilatation is secondary to that.

The exact etiology of spasm is still in doubt. Some believe the spasm is in the muscles of the diaphragm (Jackson²²), others that it is in the stomach (Fleiner), while still others believe it to be in the esophagus itself at the level of the diaphragm and not at the anatomic cardia (Abed¹⁹). Most agree that it is a true neuropathic manifestation.

The vagus and sympathetic nerves control the lower esophagus. The vagus supplies the longitudinal fibers and the tone of the sphincters. Stimulation of the vagus produces opening of the cardia and stimulation of the sympathetic produces contraction of the sphincters. Langly has shown experimentally that division of the vagi paralyzes the longitudinal fibers. Cannon, by feeding after such an experiment, has produced dilatation of the esophagus with muscle hypertrophy. Others have shown a change in Auerbach's plexus in cases of cardiospasm. Wilms suggests spasmophilia as the underlying cause. Kure, Fugii and Kawayuzi²⁰ observed by X-ray the synergic action of the sympathetic and parasympathetic on the esophagus of dogs. Complete closure of the cardia resulted when the vagi were cut. Extirpation of the superior cervical ganglion causes peculiar but mild disturbance in the upper esophagus. These authors are of the opinion that idiopathic dilatation of the esophagus is due to paralysis of the vagi which might be due to birth injury.

I have three cases of congenital cardiospasm to report:

Case 1.—C. S., age eight. Since birth had trouble swallowing. All sorts of feeding mixtures were tried, with little success during infancy. At two years of age a pyloric stenosis developed; the Ramstad operation was performed. Since then the child has been unable to take solid food. Because of hemorrhages from the bowel she was put on ulcer management and given several transfusions. Recently she had a

tooth extracted and following this was unable to swallow anything and a gastrostomy was done. I first saw the child at this time. X-ray showed an obstruction in the esophagus at the cardiac end, typical of cardiospasm. Under rectal ether an esophagoscopy was done and no obstruction of organic nature was found. A large bougie was passed easily into the stomach and left in place for three-fourths of an hour. Following this, the child was able to swallow semisolids for the first time in her life. The gastrostomy tube was removed in a few days and she left the hospital. Her progress during the six months since I dilated her esophagus has been satisfactory.

Case 2.—J. S., age one year. Since birth has had difficulty in swallowing, unable to swallow any solids. Feeding formulas have not helped. Fluoroscopy revealed an obstruction at the lower end of the esophagus with a large dilatation above. A thread was swallowed and a seven millimeter olive passed into the stomach, using chloral by rectum as an anesthetic. The child improved since this procedure, but vomits at times and is a "breath holder." Further dilatations will be necessary.

Case 3.—N. J. M., age fifteen and a half months. Since birth had difficulty in taking food—never seemed hungry. Always took an abnormally small amount of food and never any solids. Bowels were loose; forceful vomiting at times. X-ray diagnosis of enlarged thymus and X-ray treatments were given. This improved the symptoms of pylorospasm. Much atropin was given. X-ray examination at fifteen months showed a typical cardiospasm. Esophagoscopy showed no organic obstruction in the esophagus. A large bougie was passed and following this the child left the city and no further report has been received.

E. Diverticula.—The traction type of diverticulum, occurring in the lower portion of the esophagus, is rarely diagnosed as congenital. Lewis in Keibel and Mall¹⁴ states it never occurs at birth. Stan¹⁵ reports a case in a six months old baby. There was difficulty in swallowing, and the autopsy showed the lower end of the esophagus adherent to the lung tip. A pouch was formed by this adhesion. The cause of the

adhesion was thought to be pleurisy. Zenker and Peters each report one case under two years of age (Abel¹⁹). Probably none of these cases should be considered as truly congenital.

The pulsion type of diverticula may occur congenitally in the esophagus proper; Kurtz reports a case at the age of three. This may be reversion to the primitive fore stomach of certain lower animals or due to imperfect closure of a branchial cleft. (Cautley¹⁵.)

The pharyngeal pouch, seen in adults frequently, may occur as a congenital anomaly, though it is of no embryologic significance.¹⁴ Possibly it is related to the pouch found normally in the hog and other animals. The covering of this pouch has the complete muscular layers of the esophagus and is not a hernia of the mucous membrane. Judd and Mayo²¹ report a case of diverticula due to congenital stricture which was cured by dilatation. Jackson²² reports a congenital diverticulum of this type.

Case 4.—Reported by Kennedy, Phelps and Gates.⁹³ This patient was operated upon for a pharyngeal diverticula at the age of eighty. He gave a history of having had symptoms most of his life. As a boy, he would frequently regurgitate undigested food, which he had eaten several days before. For this reason the case was thought to be congenital in origin and included in this paper.

F. Esophagotracheal Fistula.—This congenital anomaly is a fairly frequent one. Some authors^{23 24} go so far as to state that, for practical purposes, all congenital esophageal anomalies are of this type. In the same article, however, they quote several authors as stating that this condition occurs in only 70 per cent of all cases.

There may be a fistula between the esophagus and trachea, with these structures otherwise normal.¹⁵ Such a condition is certainly rare, few cases being recorded.

The esophagus may end in a blind sac, which may or may not be connected with the stomach by a solid cord, often so small that it is hard to find. The atresia always occurs at a level above the bifurcation of the trachea.²⁹ The blind sac is usually dilated, perhaps on account of the fetus having swallowed amniotic fluid (Hirsch²⁵). The lower end of the esoph-

agus connects with the stomach in a normal manner, and the upper end of the lower segment opens into the trachea or a bronchus. At times there is a fistula between both segments of the esophagus and the trachea.²⁰ (Ellerbroek²⁷.)

All of these anomalies are due to a malformation of the esophagotracheal septum. The number of identical cases recorded is good evidence that this condition is due to faulty development and not due to inflammation, disease, malformation of the aorta or any other cause. The exact embryologic fault has been explained in various ways by different authors. Shattock²⁸ believes that when the lower air passages develop, from the anterior wall of the mesenteron, the posterior wall is pulled forward, and when the larynx develops later it uses up the narrow lumen and the lower segment of the esophagus is left connecting with the air passages. Keibel and Mall¹⁴ state the lower septum between the trachea and esophagus fails to develop.

Zeit²⁹ points out that this explains the fistula but not the occlusion of the upper segment of the esophagus, particularly as the esophagus is developed before the trachea and is always open. He explains the anomaly by a faulty anlage in the lower portion of the septum, causing it to grow in the wrong plane, thus joining the anterior and posterior walls instead of the lateral. This explanation would seem to be the best and most logical yet advanced. McClellan and Elterich³² think the respiratory system would develop dorsally, but, due to the spinal column, it probably follows the line of least resistance and goes anteriorly into the body cavity.

The clinical picture presented by an infant with this anomaly is typical, once seen or thought of should be easily diagnosed. The child has no trouble till he is fed, though a careful observer may notice a flow of saliva from the corner of his mouth. At feeding he will take two or three swallows and the fluid will be seen to return alongside the nipple. As the pharynx is full of fluid, the child cannot breathe; he turns blue and coughs; he is drowning. If the fluid is removed from his pharynx he again breathes freely. On examining the throat, it is seen to be full of milk and mucus, which is pathognomonic of occlusion of the esophagus (Göppert³³).

The fact that the trachea is connected to the esophagus lets some fluid out that way and drowning does not occur. Brown³⁴ reports a case with barium in the stomach which must have gone through the fistula. These symptoms recur at every feeding. Breathing is usually free between times. Meconium is passed normally. Air is seen in the stomach by X-ray. In two cases the anomaly occurred in the second twin born (Elterich³⁵ and Ellerbroek³⁷). Naltman³⁶ reports a curious case, which had varices in the blind sac, producing a clinical picture of *melena neonatorum vera*.

A catheter is found to meet with obstruction high in the esophagus. X-ray with a barium mixture shows the esophagus to be occluded at a level slightly above the bifurcation. Esophagoscopy shows the blind pouch, usually considerably dilated, but not always (Crowdy^{36, 37}).

Frequently there is some other congenital defect present, as imperforate anus, kidney anomalies, palate maldevelopment, etc.

Gastrostomy has been done on many of these infants, but no cure has been reported. Jejunostomy has been done without success (Jones and Manning³⁸). Richter³⁹ reports two cases operated by transthoracic means and closing the fistula between the trachea and esophagus and feeding the child through a gastrostomy. Both patients died. Smith⁴⁰ reports a case on which he did a gastrostomy and ligated the lower end of the esophagus. Some improvement in surgical technic may some day show us the way to successfully treat this condition.

In the literature there is some confusion as to the exact number of such cases recorded. Hirsch²⁵ states that up to July, 1920, there were 146 cases on record of esophageal atresia with 103, or 75 per cent, of the fistula type with the upper portion of the esophagus ending in a blind sac. At another point he mentions ninety-six out of 136 cases being of this type. Vinson⁴¹ adds another case and calls it 147, and Maes⁴² still another, calling his 148. There are many such cases recorded since 1920 when Hirsch's article appeared. I have been able to find twenty-seven in the literature as follows: 43, 44, 45, 46, 47, 48, 49, 25, 50, 3, 56, 57, 51, 52, 6, 30, 53, 26, 31, 54, 55, 29, 35, 58, 59, 84, 88.

Many statements are made concerning this condition which should be corrected. Zeit²⁹ found in 1912 only five such cases without other congenital defects. There are many more than that now on record. Heatly⁶² states no child has lived beyond fourteen days. I have a case which lived eighteen days. Marsh¹⁶ found no two cases in the same family, but Herzfeld²⁸ reports such an instance. Shattock²⁸ states that theoretically there might be a stricture of the trachea accompanying the fistula, but it never occurs, while Finkelstone and Ellis³⁰ report such a case and Thorek³¹ an additional one.

I have four such cases to add to the above:

Case 1.—Baby boy, Atwood Thorene, May 6, 1916, A 16-175 University Hospital. Unable to retain water or food since birth; seemed hungry but after swallowing choked and regurgitated food and a frothy mucus. Parents, delivery and external appearance normal.

Seen on fifth day by Dr. F. W. Schultz, who found a catheter met with obstruction in the upper part of the esophagus. X-ray showed a congenital atresia. No surgeon wanted to operate; the baby died on the twelfth day.

Postmortem: Both feet turned inward at ankle at right angle to leg. Foramen ovale patent.

Lungs: Pus escaped from lung on pressure, especially right.

Esophagus: Upper portion was blind sac, twenty-seven millimeters circumference at lowest extremity and seventeen at upper. Extended four and five-tenths centimeters below upper border of epiglottis. Muscle layers of esophagus end at lower border of sac, though an indefinite connective tissue bunch arises from connective tissue outside muscle wall extending down further and finally being lost in the tissue of the posterior mediastinum. Extending up from the cardiac end of the stomach was a tube corresponding to the lower end of the esophagus, which opened into the posterior wall of the trachea one centimeter above bifurcation. Lumen one centimeter in size. Intestines empty aside from gas and small amount of brown black substance and mucus.

Diagnosis: Double talipes varus; bronchopneumonia; congenital occlusion of esophagus with esophagotracheal fistula.

Case 2.—Boy, triplet, University Hospital, No. 29414-29423, A 24-403. Born May 24, 1924; mother sixteen years old, with mumps at time of delivery. Weight 2120 gms. at birth and lost weight continuously till at death weighed 1670 gms. Fed twenty cc. breast milk every three hours.

Examination: Indentation on zygomatic process. Coarse rales over left side, thought to be mucus in bronchi. Four days later noted child did not retain any of its feedings. Barium meal showed esophageal stenosis, opposite third thoracic vertebrae. Fifth day, gastrostomy done; retained food. June 4th. noted brown discharge from mouth and child died.

Postmortem: Ductus arteriosus and foramen ovale are open but compensated.

Lungs: Putrefactive odor and yellow material from bronchi.

Esophagus: Dilated from pharynx to bifurcation of trachea, where it ends in a pouch. Just below the end of this pouch is an opening from anterior wall of esophagus into trachea, and below here the esophagus is of normal size.

Diagnosis: Congenital occlusion of the esophagus; esophago-tracheal fistula.

Case 3.—Baby boy Wheeler, University Hospital No. 28611. This child was brought to the hospital after having the diagnosis of esophagotracheal fistula made on the outside and a gastrostomy had been done. The diagnosis was confirmed and no further treatment was attempted. Death occurred on the eighteenth day. No postmortem was permitted.

Case 4.—Baby boy born at University Hospital August 22, 1928. Admitted to nursery with poor color, marked dyspnea, large amount of mucus in throat and with very evident obstruction to respiration. It was necessary for the infant to use all the accessory muscles. The trachea was milked and a moderate amount of blood tinged sputum was brought up. Oxygen seemed to bring the color back. Attacks every five minutes, then half hour intervals and ended spontaneously by bringing up mucus which contained less blood. Then condition grew worse and oxygen was used again.

Diagnosis of thymic stridor was made. X-ray examination showed only moderate enlargement of the thymus. There was

marked displacement of the mediastinum and heart into the left chest. The heart seemed somewhat enlarged. The entire picture resembled somewhat an abnormality of the relation of the heart to the thorax. The larynx was examined during a paroxysm and the cords were found approximated. Fluids by bowel and in the peritoneum were given. An attempt was made to give water by lavage but the tube was arrested before it was thought to enter the stomach and the water was returned almost immediately.

Postmortem: There was air in the stomach. Examination of the mediastinum revealed a complete atresia of the esophagus with marked dilatation of the segment above. There was a communication between the upper part of the lower esophageal segment and the bifurcation of the trachea.

The scalp showed hemorrhage and edema. There was marked hemorrhage of the pia arachnoid and a large amount of free blood in the base of the skull. Deep bilateral tears and hemorrhages of the tentorium. Multiple hemorrhages of the falx. The brain showed congestion and softening.

Diagnosis: Birth trauma (laceration and hemorrhages of tentorium); hemorrhages of pia arachnoid and cerebrospinal fluid; partial pulmonary atelectasis; congestion of viscera; anomaly of thumb; uric acid infarcts of kidneys; atresia of esophagus; esophagotracheal fistula.

G. Cysts of the esophagus occur as congenital anomalies but are exceedingly rare. Bittenwieser⁶⁰ reports such a case. There was a cyst on the anterior and right wall of the esophagus the size of a pigeon's egg. It was found at autopsy on a seven day old child. No other esophageal anomaly was found. Cantley¹⁵ states the cyst may arise during the separation of the trachea and esophagus, and refers to a case of Hibb in a woman thirty-one years old. In *Abt System*, Vol. III, page 409 (61), Heuman states fifteen cases collected by Bittenwieser, thirteen without symptoms and found at autopsy.

H. Atresia of the esophagus is due to one of the following developmental errors:

(a) The early esophagus, referred to as the sphincter in the four millimeter embryo, may not elongate equally and thus leave a stenosis in a portion of the canal.

(b) The stage of obliteration, which is never complete, may fail to open up as wide as it should.

(c) The vacuolization of the cells is incomplete or the vacuoles fail to coalesce.

Atresia may occur at the cardiac end of the esophagus, as reported by Marcus,⁶³ in which case the entire esophageal wall seemed to take part in the stenosis. Abel⁶⁴ reports a case with a complete web across the esophagus at one inch below the level of the bifurcation. Esophagoscopy was done at two days of age; the web was perforated and the child lived. Fitzgibbon⁶⁷ reports another such case. These cases are unique in the literature.

Many cases in which the middle third of the esophagus is replaced by a solid cord are on record. The upper end of the atresia is at the level of the cricoid and a solid cord extends to the cardiac end of the esophagus, which opens normally to the stomach (25, 65, 17, 66, 38, 85, 30, 42, 6, 3, 91).

This condition is allied to malformation of the esophago-tracheal septum (Kieth¹¹) and associated with the elongation of the esophagus which occurs during the development of the lungs. Crowdy³⁶ found a ridge at a point on the posterior tracheal wall opposite the blind esophageal pouch.

I. Stenosis or incomplete occlusion is of more frequent occurrence. Griffith and Lavenson⁷⁶ found twenty cases reported up to 1909, while Hofer,⁷⁰ in 1928, reports a total of forty-five. It may occur at any level in the esophagus. Morse⁸⁰ reports a case at six inches from the incisors, and Keith¹¹ reports nine specimens in the Royal College of Surgeons (London), in which there is a stenosis of the upper orifice. It is not a hypertrophy of the constrictors, but a true fibrous stenosis. The circular muscle is replaced by loose connective tissue.

Heatley,⁶² Morse⁸⁰ and Grieg⁷⁸ report cases of stenosis of the middle third, while Hutchison⁷⁹ has a case of stenosis one and a half inches below the bifurcation, and Fitzgibbon⁶⁷ a case ten inches from the incisors.

Turner,⁸¹ Mayer⁷⁷ and Eschelman⁶⁹ report cases of stenosis at the lower end. Vinson⁴¹ has four cases at various levels.

Krokiewicz⁹⁰ has a case of stenosis of both the esophagus and pylorus with subsequent dilatation of each (gastrectaseia).

Others^{67 82 73 80} report cases at different levels, and Arens and Bloom⁶⁸ have a case of stenosis at two separate points. Frey⁷² found a valve-like protrusion, one to two centimeters from the cardia. This he cut and dilated.

Cases are reported as congenital in patients ranging in age from a few days to sixty-seven years.

Most authors believe that the entire wall of the esophagus takes part in the stenosis, and many report good results by dilatation, carried on by various methods, such as the olive tip bougie on a thread (Plummer), bougies of the Jackson type or others or retrograde. This procedure must be carried on for a varying length of time.

Whipham and Fagge⁷³ report a case of ruptured esophagus following dilatation and state six others are in the literature.

Other authors^{74 78} believe diet is the only satisfactory treatment.

Guisez⁸¹ reports a case, the first symptom of which was a foreign body lodging in the congenitally narrow esophagus.

I have a case of partial occlusion of the cardiac end of the esophagus to report:

Baby Remillard, age seven weeks, was referred to me by Dr. E. D. Anderson because of choking and cyanosis which occurred at each feeding. There seemed to be an unusual amount of mucus in the baby's throat, which at times necessitated removal by means of a catheter and suction.

The previous history is as follows: At two weeks of age the baby vomited its food. The vomiting was projectile in character and occurred after one or two feedings a day. This became worse, and at three and a half weeks the baby vomited nearly everything. She lost weight rapidly. She was then taken to the hospital and fed breast milk per tube. Tubing was difficult and seemed to make her vomiting worse than ever. Atropin in large doses produced no improvement. X-ray showed 90 per cent of the gastric contents passed through the pylorus at the end of four hours. The diet was then changed to thick cereal, and she could keep this down, though it took nearly an hour for her to take two ounces. Water made her vomit at

once. Hypodermoclysis was given daily to keep up the fluid intake.

At five weeks she began to have choking attacks and cyanosis, which was much worse during feedings. X-rays were again taken, which showed definite stenosis of the lower end of the esophagus with considerable dilatation above. (See illustration.)

The esophagus was dilated by the Plummer method and the baby immediately improved. She was able to take liquids, so hypodermoclysis was discontinued. She gained weight at once. Dilatation was repeated four times and she left the hospital in good shape. Six months later dilatation was repeated. One year later repeated and again two years later. She is now five years old and can swallow as well as her twin sister. X-ray shows no obstruction in the esophagus.

Von Gilse⁸³ reports a case he considers due to paralysis caused by birth injury. This is the only case I have located in the literature.

Stenosis caused by pressure from without is also rare in the literature. Mosher^{7 8} calls attention to the action of the lung tips in causing compression stenosis at the lower end. Straus and Hess⁷¹ report cases caused by connective tissue bands around the esophagus from the diaphragm and describes an operation to cure the condition by cutting the bands. They had three cases and all died of pneumonia following the operation. These authors state that stenosis at the cardiac end is rare and is never complete.⁶³ They attribute some of the obstruction to spasm superadded to the stenosis. Peterson⁹⁴ is quoted, who describes an operation for congenital stenosis which he did on a seventeen year old girl, making an esophagus subcutaneously.

CONCLUSION.

1. Congenital anomalies of the esophagus are not rare.
2. There are nine definite varieties of anomalies.
3. Certain types can be treated successfully; hence an accurate diagnosis should be made in every case.

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BIBLIOGRAPHY.

1. Kreuter: Habildationschrift. Erlangen, 1905.
2. Plass: Johns Hopkins Hospital Report 1919, 259.
3. Reynolds and Morrison. Am. J. Dis. Child., 1921, 339.
4. Vinson. ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1927, XXXVI, 40.
5. Stukowsky and Boran: Arch. f. Kind., 1912, LVIII, 119.
6. Neff: Am. J. Dis. Child., 1921, XXII, 57.
7. Mosher: ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1926, XXXV, 969.
8. Mosher: ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1928, XXXVII, 12.
9. Stam: Monat. f. Kinderheil., 1926, XXXIII, 147.
10. Losee: Bulletin Lying-In Hosp., N. Y., 1924, January.
11. Kieth: B. M. J., 1910, I, 300.
12. Johnson, F. P.: Am. Jour. Anat., 1910, X, 521.
13. Reese: Am. Jour. Anat., 1926, XXXVII, 195.
14. Keibel and Mall: Embryology, II.
15. Cautley: B. J. Child. Dis., 1917, XIV, 1.
16. Marsh: Am. J. Med. Sc., 1902, CXXIV, 304.
17. Parrish: Jour. A. M. A., XXXVI, No. 1, 33.
18. Moersch: Am. J. Dis. Child., 1929, XXVIII, 294.
19. Abed: Esophageal Obstruction. Oxford Press, 1929. Quoted frequently throughout this paper.
20. Kure-Feyii and Kawayuzi: Klin. Woch., Berlin, 1929, VIII, 491.
21. Mayo, C. H.: Ann. Surg., 1923, LXXVII, 267.
22. Jackson: Ann. Surg., 1926, LXXXIII, I, 19.
23. Brenneman: Am. Jour. Dis. Child., 1918, XVI, 143.
24. Brenneman: Am. Jour. Dis. Child., 1913, V, 143.
25. Hirsch: Jour. A. M. A., 1921, LXXXVI, 1491.
26. Willard: Jour. A. M. A., 1922, LXXVIII, 649.
27. Ellerbroek: München Med. Wochenschr., 1922, LXIX, 591.
28. Shattock: Trans. Path. Soc. London, 1890, XVI, 87.
29. Zeit: Journal Med. Research, 1912, XXII, 45.
30. Finklestone and Ellis: Jour. A. M. A., LXV, 2155.
31. Thorek: Jour. A. M. A., XXVI, No. 1, 33.
32. Kieth and Spicer: Jour. Anat. and Phys., 1907, XLI, 52.
33. Göppert: München, Med. Wochschr., 1921, LXVIII, 1649.
34. Brown: Radiology, 1926, VII, 166.
35. Elterick: Am. Jour. Dis. Child., 1923, XXVI, 373.
36. Crowdy: Jour. Med. Research, 1918, XXXVIII, 409.
37. Crowdy: Jour. Med. Research, 1917, XXVIII, 469.
38. Jones and Manning: Jour. A. M. A., 1916, LXVI, March 11.
39. Richter: Surg., Gyn. and Ob., 1913, XVII, 397.
40. Smith: Am. Jour. Surg., 1923, XXXVII, 157.

41. Vinson: Jour. A. M. A., 1923, 1754.
42. Maes: Am. Jour. Surg., 1926, I, 153.
43. Kipper: Med. Klin. Berlin and Wien, 1927, XXXIII, 1377.
44. Taglicht-Virchow: Arch. f. Path. Anat., 1921, CCXXIX, 322.
45. Zausch-Virchow: Arch. f. Path. Anat., 1921, CCXXXIV, 94.
46. Theron: B. M. J., 1926, I, 652.
47. Schiro: Cal. and West. Med., 1926, XXIV, 505.
48. Litchfield: Arch. Ped., 1927, XLIV, 310.
49. Knerr: Radiology, 1925, V, 165.
50. Holderman: Arch. Surg., 1927, XIV, 917.
51. Steffen: Arch. Ped., 1922, XXXIX, 823.
52. Flood: Atl. Med. Jour., 1926, XXIX, 538.
53. Weiss: Jour. A. M. A., 1923, LXXX, 17.
54. Shaw: Am. Jour. Dis. Child, 1920, XX, 507.
55. Kostner: Arch. Ped., 1920, XXXVII, 11.
56. Huntington, Young and Foot: Boston M. and S. J., 1919, CLXXX, 354.
57. Kas and Avery: Iowa State Med. Soc., 1923, XIII, 275.
58. Herzfeld: Zentralbl. f. Gynäk., Leipzig, 1926, I, 3076.
59. Smith, G. E.: Can. M. Monthly, 1920, V, 219.
60. Buttenwieser: Ztsch. f. Kinderh., Berlin, 1922, XXXII, 352.
61. Abt: System of Ped., III, 409.
62. Heatley: Arch. Otol., 1928, VIII, No. 1, 66.
63. Marcus: N. Y. M. J., 1923, CXVIII, 374.
64. Abel: B. M. J., 1928, II, 46.
65. Skinner: A. M. J. of Roentgen., 1921, VIII, 319.
66. McLoone: Wis. M. J., 1927, XXVI, 258.
67. Fitzgibbon: N. W. Med., 1926, XXV, 94.
68. Arens and Bloom: Radiology, 1926, VI, 163.
69. Eschelman: Bull. Buffalo Gen. Hosp., 1923, I, 24.
70. Hofer: Monat. f. Ohrenheil., April, 1928.
71. Straus and Hess: Jour. A. M. A., LXXXIV, 501.
72. Frey: Abstract. Jour. A. M. A., 1922, LXXIX, 43.
73. Apfal: N. Y. Med. J., 1918, CVIII, No. 3, 108.
74. Brown, K. P.: Edinburgh Med. Jour., 1922, XXIX, 145.
75. Whipham and Fagge: Lancet, 1905, I, 22.
76. Griffith and Lavenson, Arch. Ped., 1909, XXVI, 161.
77. Mayer: Am. Jour. Med. Sc., 1893, CVI, 567.
78. Grieg: Edinburgh Med. Jour., 1921, XXVI, 342.
79. Hutchison: Proc. R. Med. Soc., 1923, XVI. Children's Section, 42.
80. Morse: Amer. Jour. Dis. Child., 1920, II, 144.
81. Turner: Trans. Path. Soc. London, 1885, XXXVI, 185.
82. Imperialè: Riforma Med. Napoli, 1927, XLIII, 721.
83. Von Gilse: Ztsch. f. Hals u. Ohrenheil., 1928, XXII, 91.
84. Simpson: Arch. Oto. Lar., 1929, III, 267.

85. Sanders: B. M. J., 1926, II, 938.
86. Noltman: Ztsch. f. Geburtsch. u. Gynäk., 1926, XC, 260.
87. Guisez: Bull. d'oto-rhino-laryngol., Paris, 1927, XXV, 333.
88. Rhenter, Pigeaud and Levert: Bull. Soc. d'obst et de gynec, 1928, XVII, 403.
89. Kraes: Beitr. z. Klin. Chir. Tubing, 1927, CXLI, 281.
90. Krokiewicz-Virchow: Arch. f. Path. Anat., 1926, CCLIX, 761.
91. Kotzareff: Ann. de gynec. et d'obst., 1918, XIII, 203.
92. McClellan and Elterich: Am. Jour. Dis. Child.
93. Kennedy, Phelps and Gates: Minn. Med., 1928, October, 669.
94. Peterson, O. H.: Beitr. z. klin. Chir., 1921, CXXIV, 705.
95. Birnberg, T. L.: Am. Jour. Dis. Children, 1929, Dec. Vol. 38, p. 1183.

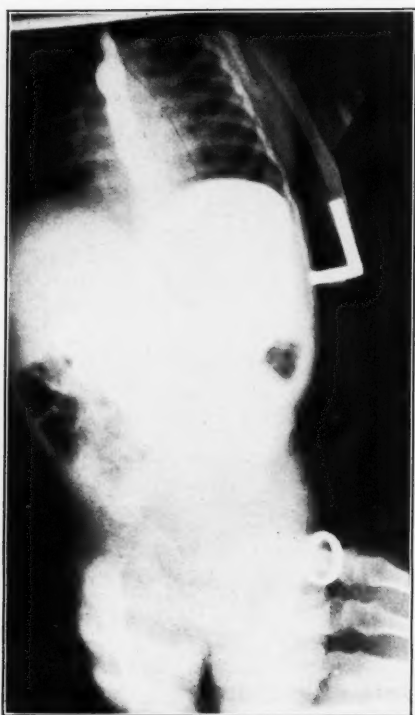


Fig. 1. Very narrow stream of barium enters stomach through lower end of esophagus with moderate dilatation of esophagus above.

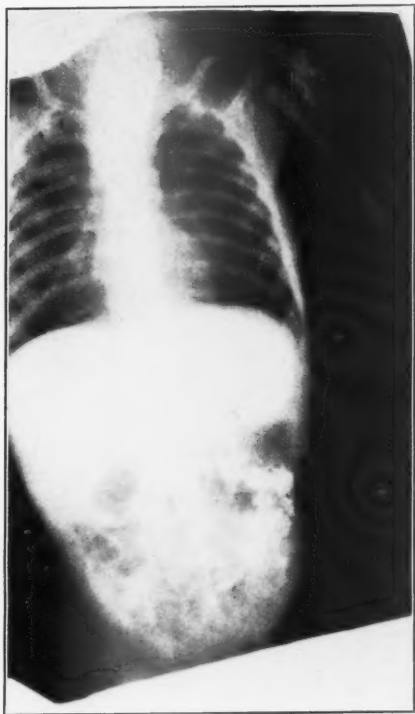


Fig. 2. Taken a few days later, shows how the dilatation has increased enormously. The stenosis of the cardiac end of esophagus is nearly complete.

XXVI.

THE SURGICAL ANATOMY OF THE LYMPHATICS
OF THE HEAD AND NECK.*

By HOMER A. TROTTER, M. D.,†

BUFFALO.

The lymph glands, or more appropriately, the lymph nodes, are structurally composed of a peculiar form of cell which, when grouped together, is called lymphoid tissue. Lymph nodes are small, bean shaped organs, surrounded by a capsule and composed of cortex, medulla and hilus. The capsule consists of white fibrous tissue and contains some yellow elastic and scattered smooth muscle tissue. Beneath is a lymph space or sinus. From the inner surface of the capsule trabeculae are sent into the cortex, and these divide the latter into a number of masses called secondary follicles or nodules. The lymph space continues along the trabeculae. The cortex contains the secondary nodules and trabeculae. The former consists of dense lymphoid tissue and contain a germinal center. The cells are chiefly lymphocytes, which are arranged in concentric layers around the periphery. Other cells of the hyalin variety are found in the central portion. The follicles continue into the center of the node as the medullary cords. The trabeculae separate the follicles from one another and pass into the medulla surrounded by the lymph space.

The medulla consists of the medullary cords and trabeculae. The cords are the bandlike continuations of the secondary follicles and are separated from the trabeculae by the lymph spaces that accompany the latter. They consist of dense lymphoid tissue supported by reticulum. At the hilus the medulla comes to the surface.

The lymph node lies loosely in connective tissue and is held in place by a connective tissue reticulum. The blood supply is

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†1. P. Poirier et A. Charpy. *Traite D'Anatomie Humaine*. 2me tome, 4me fascicule, 2me edition, Paris, 1909.

received through its capsule, also the afferent lymph vessel, draining lymph from the area beyond; after passing through the node, the lymph finds its exit by way of the efferent vessel, finally reaching the blood stream through the vein into which the lymph vessel empties.

On the periphery of the follicle the lymphoid cells are smaller than in the center, in which many of the larger show karyokinesis.

The lymph in passing into the node is in direct contact with the lymphoid cells and undoubtedly altered in composition and cellular contents.

The nodes may be influenced by the blood brought to them and by the lymph of the area drained. There are about 800 lymph nodes scattered in definite areas through the body. Their distribution is significant in that provision seems to have been made for their presence in areas where infection might occur, for example, the thorax in relation with the heart and lungs; the abdomen, in connection with the gastrointestinal tract and genitourinary system; the neck which contains about 300 nodes and located to protect infections arising in the mouth, nose, sinuses, nasopharynx and about the face.

It is comparatively seldom that they are the seat of primary disease. Secondary enlargement, however, almost constantly follows infections of the areas which they guard and they form the first barrier to infections of the areas which they protect. They also form the first line trenches to infectious materials that have escaped into the lymph stream.

LYMPH NODES OF THE HEAD AND NECK.

In the cervical region are located the principal lymphatic gland development, which forms a collar or circle placed at the junction of the head and neck on each side. This group lies beneath the sterno-cleido-mastoid muscle and accompanies the large vessels and nerves to the junction of the neck and thorax. This principal chain is flanked by chains of glands of less importance.

The circle of lymph glands may be subdivided into a number of groups and named from the region occupied.

From the back to the front of the head and neck are found the following groups: Suboccipital, mastoid, parotid, submaxillary, submental and retropharyngeal.

1. The suboccipital group vary from one to three glands, usually three. They are about the size of a pea and ordinarily lie at the insertion of the occipital muscle at the outside of the border of the trapezius muscle and sometimes on the surface of this muscle. Occasionally this group may be found in the superficial aponeurosis or lie on the sheath of the suboccipital nerve.

This group receives the afferent vessels from the lower occipital part of the head, and the efferent vessels run to the upper substernal mastoid glands.

The substernal glands receive directly from the occipital region.

2. The mastoid glands (retroauricular) usually occur in pairs, always visible in children but difficult to discover in the adult. They are placed one behind the other and are united by two or three trunks. They are found at the junction of the insertion of the sterno-cleido-mastoid muscle. The mastoid glands receive afferent vessels from (a) the temporal part of the head, (b) the internal surface of the ear, and (c) from the posterior part of the auditory canal.

The efferent trunks drain the superior group of the sterno-cleido-mastoid glands.

3. The parotid group comprises the subcutaneous glands, although they are often missing; also the superficial and deep glands, over the parotid area under the superior aponeurosis. The superficial are found in the newborn and frequently in the adult, and are located at the upper surface of the gland. They are one to two in number and located in front of the auditory canal.

The deep group are disseminated throughout the parotid gland.

The afferent vessels are received from the (a) external part of the ear, (b) from the skin over the temporal and frontal regions, (c) eyelids, (d) and outer part of the nose, (e) the anterior part of the auditory canal. Perhaps receive lymph from the mucosæ of the nasal fossæ.

The efferent vessels empty themselves at the internal jugular at the outlet of the parotid and the submaxillary glands.

The number of glands of the parotid group vary in number, three to sixteen. There is a subparotid group which belongs to a chain along the route of the internal jugular. The majority of authors do not mention this group.

4. The submaxillary group varies from three to six glands and is found along the inferior border of the maxilla from the anterior insertion of the digastric muscle to the angle of the jaw. The glands are found lying upon the submaxillary gland and are about the size of a pea and are under the aponeurosis.

A posterior gland may be found behind the facial vessels at the angle of the jaw.

A middle group are most common. There are no glands found in the parenchyma of the submaxillary gland as there are in the parotid gland.

This submaxillary group receives afferent vessels from (1) nose, (2) cheek, (3) upper lip, (4) external part of inferior or lower lip, (5) all of the gum, (6) anterior part of the tongue.

The efferent vessels descend over the submaxillary gland and cross the hyoid bone to empty into the deep cervical chain, especially the glands at the bifurcation of the carotid artery. A subdivision of the submaxillary group are the facial group, which in turn is divided into (1) superior, (2) media, and (3) inferior.

The inferior is located at the external side of the inferior maxillary between the anterior border of the masseter and the posterior part of the lips. There are observed one to three glands close to the facial artery and vein.

The medial group is found deeply located, external to the buccinator muscle.

The superior group is less important than the other group and placed in the suborbital region.

5. The submental group is found in a triangle bounded by the digastric muscle and the hyoid bone. They vary in number from one to four, but are usually two, and in the adult may be entirely missing. Also missing in the newborn.

The afferent vessels are from the (1) skin over the chin, (2) middle of the lower lip, (3) mucous membrane of the

mouth in a corresponding area, (4) floor of the mouth, (5) the tip of the tongue.

Efferent vessels follow a double course, partly to the sub-maxillary glands and, by the way, toward the hyoid bone to empty into a gland located on the anterior part of the face at the external jugular vein.

3. Retropharyngeal glands are found behind the pharynx on the posterior and lateral sides at the level of the atlas bone. There are usually two glands or there may be one.

The afferent vessels are from the mucosæ of (1) the nasal fossæ, (2) the paranasal sinuses, (3) the nasopharynx, (4) eustachian tube and lymphatics from the internal ear. Because of the extended area involved in this group numerous infections are observed.

Efferent vessels empty into the internal jugular chain.

THE DESCENDING CERVICAL CHAIN.

This group of glands descend with the great vessels of the neck to the chest. They are also known as the deep cervical nodes. This group is flanked by a secondary chain of glands of less importance and called the external jugular chain.

The deep cervical chain, also called the carotid group, is found beneath the sterno-cleido-mastoid muscle and constitutes the deep glands of the neck. This chain of lymph nodes forms the most important group of lymph nodes in the whole body. There are fifteen to thirty glands found in this group.

The deep cervical chain is divided by some authors into the superior and inferior groups and so divided by the omohyoid muscle. The superior group is formed by the upper deep cervical glands. The inferior group is formed by the subclavian glands.

The deep cervical chain extends from the mastoid bone and posterior border of the digastric muscle in a vertical direction to where the omohyoid muscle crosses the deep vessels and nerves. All are covered by the sterno-cleido-mastoid muscle and are adherent to the under layer of the muscle. While this chain makes one continuous descent, they may be divided into external and internal groups, differing from the origin of their afferent vessels.

The external group is placed behind and outside of the internal jugular. They are small in size, round and occur without apparent order. This group, especially, receives the cutaneous lymph vessel of the head and neck.

The internal glands lie on the internal jugular or external border of it. The glands are more numerous than the external group and certain glands have a fixed position. They are disposed parallel to the internal jugular. Certain of these nodes have a fixed position. One or sometimes two are found at the outer border of the digastric muscle and they drain the principal region of lymphatic supply to the tongue.

Another large group lies at a point where the omohyoid muscle crosses the internal jugular vein; also, several nodes are found behind the internal jugular vein and in front of the prevertebral muscles. There is no fixed position of these glands and they are closely anastomosed by their lymphatic vessels.

The external group receives the efferent vessels of the mastoid glands, the suboccipital and some vessels from the glands about the external jugular. One large vessel comes from the occipital region of the scalp. Several trunks come from the lobe of the ear. The cutaneous lymphatics from the upper part of the neck. Some of the external group receive vessels which should go to the internal group—for example, the nasal fossæ and the nasopharynx.

The internal group receives efferent vessels from the retropharyngeal nodes, the parotid and subparotid nodes, submaxillary and submental nodes. In addition, they also receive the large part of the lymphatics from the tongue, nasopharynx and all of the middle and inferior part of the pharynx. Some from the cervical part of the esophagus, nasal fossa, cervical part of the trachea and the thyroid gland. The internal group receives from the tonsils, hard palate, orbit and the larynx.

THE ACCESSORY CERVICAL CHAIN.

The external jugular group or the accessory cervical chain of glands is formed by four or five glands and located at the external jugular vein as it leaves the parotid gland. They lie on the external surface of the sterno-cleido-mastoid muscle, a little behind the pole of the parotid gland. They receive the

afferent vessels from the lobe of the ear and parotid region. The efferent vessels are found along the anterior border of the sterno-cleido-mastoid muscle and empty into the superior glands of the deep cervical chain. One of these vessels follows the external jugular vein and empties into the subclavicular glands.

The superior, anterior cervical group is composed of two or three small glands and are not constant. They are found along the anterior jugular vein.

The deep anterior cervical chain glands are found under the subhyoid muscles in front of the laryngotracheal nerve.

The recurrent cervical chain group consists of three to four small glands placed along the side of the esophagus, larynx and trachea. They are found following the course of the recurrent laryngeal nerve. They are very small and difficult to find.

SURGICAL SIGNIFICANCE.

The retropharyngeal and cervical lymph nodes, because of the extensive area involved through afferent lymphatic vessels, are frequently the seat of numerous infections and malignant changes.

It is obvious, therefore, for the surgeon dealing with these cases to possess a knowledge of the relation with the periphery. It has been accepted by most authors that the flow of lymph is toward certain groups of lymph nodes, thus making it possible to develop a table which is a positive help to the surgeon in the eradication of diseased lymph glands. The table is practically as follows:

SUBOCCIPITAL GROUP.

- (a) Lower occipital area of scalp.

MASTOID GROUP.

- (a) Temporal part of the head.
- (b) Internal surface of the ear.
- (c) Posterior part of the auditory canal.

PAROTID GROUP.

- (a) External part of the ear.
- (b) Skin from temporal and frontal area.

- (c) Eyelids.
- (d) Outer part of nose.
- (e) Anterior part of auditory canal.
- (f) Occasional vessel from mucous membrane of the nose.

SUBMAXILLARY GROUP.

- (a) Lower nasal fossæ.
- (b) Buccal cavity.
- (c) Upper lip.
- (d) External part of lip (lower).
- (e) All of the gums.
- (f) Anterior part of tongue.

SUBMENTAL GROUP.

- (a) Skin over chin.
- (b) Middle of lower lip.
- (c) Mucous membrane of mouth.
- (d) Floor of mouth.
- (e) Tip of tongue.

RETROPHARYNGEAL GROUP.

- (a) Nasal fossæ.
- (b) Paranasal sinuses.
- (c) Nasopharynx.
- (d) Eustachian tube.
- (e) Vessels from the internal ear.

THE DEEP CERVICAL CHAIN.

EXTERNAL CERVICAL GROUP.

- (a) Cutaneous vessels from the head and neck.
- (b) Efferent vessels from mastoid and suboccipital nodes.
- (c) Occipital region of the scalp.
- (d) Lobe of the ear.

INTERNAL CERVICAL GROUP.

- (a) The greater part of the tongue.
- (b) Efferent vessels from the retropharyngeal, parotid, submaxillary and submental nodes.
- (c) Posterior part of the tongue.
- (d) Nasopharynx, middle and inferior pharynx.

- (e) Trachea.
- (f) Esophagus.
- (g) Nasal fossæ.
- (h) Thyroid gland.
- (i) Tonsils.
- (j) Roof of the mouth.
- (k) Orbit.
- (l) Larynx.

There are exceptions to the usual route of the lymphatic vessels; for instance, in some cases lymphatics from the right side of the tongue pass to nodes in the left side of the neck, and instances of anomaly occur elsewhere.

It has been observed after the removal of a group of lymph nodes that the area drained by their tributaries become involved in a swelling, persistent and indurated, and known as hard edema of lymph edema.

Collateral lymphatic circulation is established when the vessels are damaged by trauma or disease.

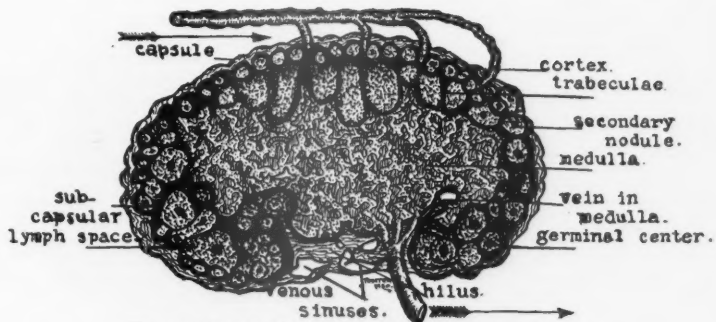


Fig:1. Longitudinal Section of a Lymph Node.

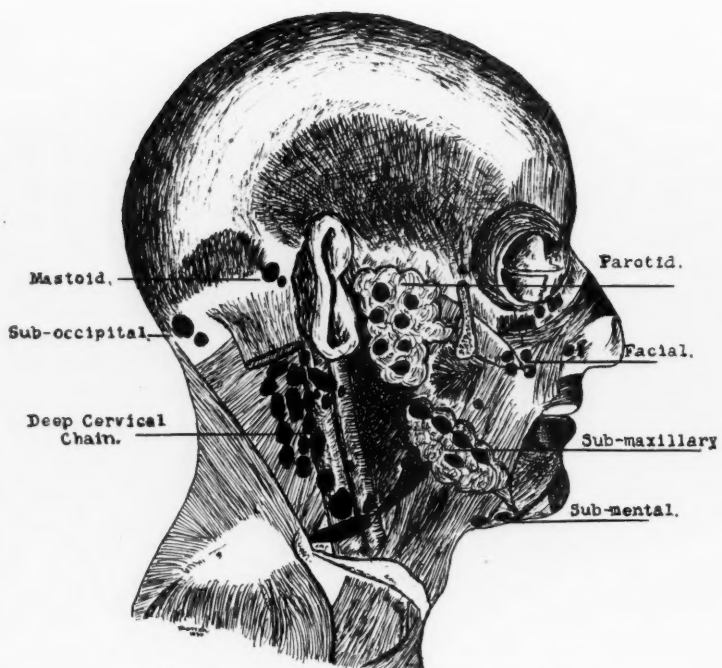


Fig-2.. General arrangement of the lymph nodes of the head and neck.

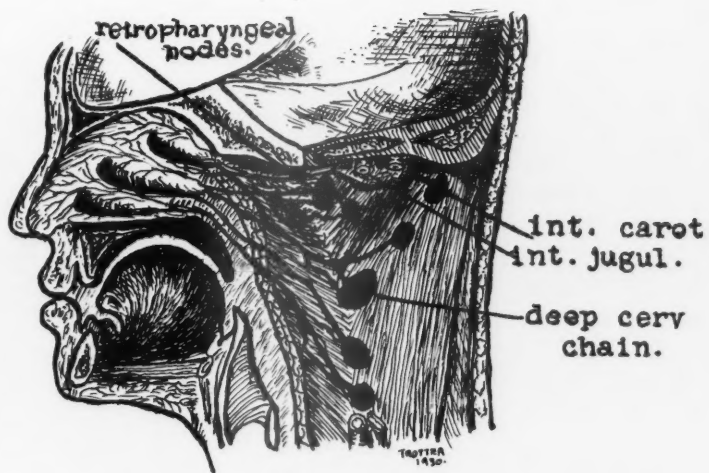


Fig:3.Lymphatics of the external
part of the nasal fossae.

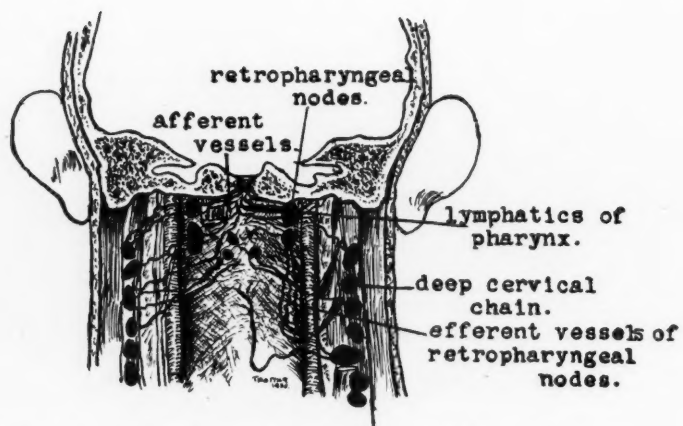


Fig:4 The Retropharyngeal Nodes. (Poirier)

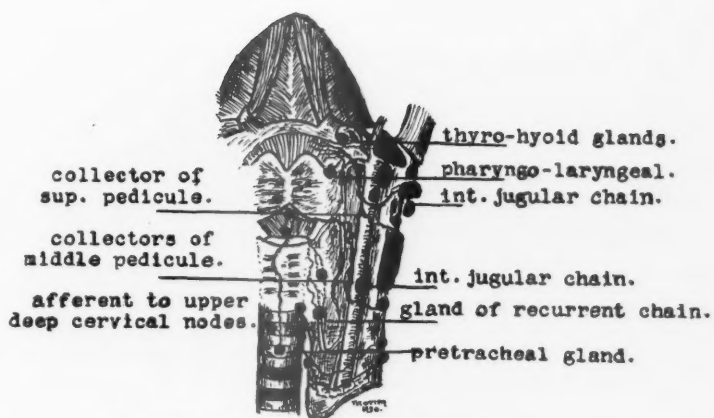


Fig:5. Lymphatics of the Larynx.

XXVII.

AN ENORMOUS FRONTAL LOBE ABSCESS FOLLOWING INFECTION OF THE FRONTAL SINUS: OPERATION, RECOVERY.

By

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PHILADELPHIA.

It was just thirty years ago that the first report of a recovery of frontal lobe abscess following operation was published,¹ and it was looked upon then as unique and not apt to occur again in the near future. Since that time and up to the present, we have been able to collect about 177 cases of frontal lobe abscess, most of which had undergone operation, with a mortality of approximately 75 per cent and which would place the number of recoveries at about 44.

This high mortality was due to the fact that these abscesses were situated in the silent area of the brain and many remained unrecognized (about 45 per cent) until death occurred and an autopsy was made. As late as 1924, one of us (Skillern) made the statement that he did not recall a case where the operator opened up the frontal sinus and drained a blind abscess in the frontal lobe that was diagnosed primarily.² This holds good almost up to today, for if we carefully review the literature we will note that almost every case had undergone one or more previous operations for frontal sinusitis when the brain abscess certainly had been present.

In making this statement we refer, of course, to those cases which were due to a previous frontal sinus infection. Eagleton,³ in 1922, reported 150 cases of frontal lobe abscess, 64 of which certainly took their origin from the frontal sinus. We have been able to augment these by 27, so that the total number to date counts 92, which is about one-half of the 177 reported. While the mortality following operation is still very high (about 70 per cent), it is steadily becoming lower, due to the improvement in surgical methods made by such men as Els-

berg, Kerr and particularly King,⁴ even though the latter, in our opinion, has gone a trifle too far in his radicalistic tendencies. The cause of this high mortality, according to Eagle-ton,⁵ who, in 1925, said it was over 80 per cent, was largely due to imperfect methods of treatment. It is on this very account that we are reporting our last case and discussing in some detail the after-treatments which have proven most efficacious in our hands.

This case is particularly striking, from a number of stand-points, the most interesting being her serious condition immediately preceding operation and the almost instant improvement with uneventful recovery after the abscess had been located and drained.

L. W., 20 years old, West Chester, Pa.—Previous history as to her local disease negative, except that at eleven years of age she was kicked in the right frontal region by a horse. There was apparently no concussion and no fracture, but a large skin flap just above the hair line was raised. This was sutured and healed readily with little swelling. A scar is still evident when the hair is parted.

Present illness began December 8, 1929, with a cold in the head, soon followed by fairly severe right frontal headache. On the 9th of December she rode horseback for several hours on a raw, cold day. On her return, headache was worse and remained so. Dr. Klevan of West Chester, was called in and found her right frontal region tender, the right nasal mucosa swollen and pus in the right nostril. He treated this by ephedrine packs, followed by neosilvol.

She was first seen by one of us (Dr. Coates) on December 11th, in consultation. An X-ray taken at that time showed a somewhat cloudy right frontal sinus. Examination revealed an engorged mucous membrane, right middle turbinate hypertrophied and swollen, and crowded between the septum and the lateral nasal wall, much thick mucopus in the nostril and, after cleansing, pus was seen coming from beneath the anterior end of the right middle turbinate. She had a massive edema of the right upper eyelid which could not be raised. The forehead also was edematous above the brow, more marked on the right but extending across the midline to the left.

Tenderness was marked. Shrinking and suction produced a considerable amount of pus with some relief of the constant headache. Her temperature had been fairly steady between 100 and 102, with pulse around 100. She complained a good deal of headache but had no other symptoms of note.

She was seen again on the 15th, at which time slight improvement was shown, the edema being less and the pain considerably improved. The bacteriologic examination revealed pure culture of staphylococcus albus hemolyticus. Dr. Klevan had given her one dose of eolan and prepared a vaccine from the staphylococcus organism.

On December 18th, the condition being in no wise improved, the anterior end of the middle turbinate was removed under local anesthesia. As there was distinct evidence of pus under the upper eyelid below the middle of the brow, this was incised and a small amount of thick white pus evacuated at a depth of about one-half inch. The organism was again staphylococcus hemolyticus. No fistula into the frontal sinus was discovered and no necrotic bone felt with a probe. Dr. Klevan reported that following these procedures, during the course of the next three weeks, the nose drained freely for a while, later ceasing. The opening through the upper eyelid drained for three days and then closed. The edema of the upper eyelid disappeared almost entirely, as also the edema over the forehead. The headaches, however, continued and there was some photophobia. The temperature was normal after about December 25th. However, she did not do well, gradually losing strength and complaining of constant and increasing frontal headache which required morphin for its control.

On January 8th, her condition became much worse. She was markedly weaker and had a subnormal temperature with a pulse at times below 50. On January 10th, conditions became so alarming that she was brought in an ambulance from West Chester to the Graduate Hospital in Philadelphia, a distance of thirty miles. She had to be assisted downstairs into the ambulance and is said to have fainted on the way down. One-half hour after admission she became semicomatose, being aroused with the greatest of difficulty and only after hesitation answering questions, *yes* or *no*. In another half hour she developed

a complete hemiplegia on the left side, with some weakness of the left facial muscles.

Previous History.—Has had measles, pertussis, mild influenza in the spring of 1929. Patient was not a strong child, but after her thirteenth birthday she put on weight and seemed healthy in every way until 1926, when she had amenorrhea for ten months. Tonsils and adenoids were removed in 1920.

Physical Examination.—A well developed and a fairly well nourished, pale, white female, about twenty years old, who was first seen lying on her right side in a stuporous condition with eyes closed and breathing about 15 times per minute. The head was drawn back and the muscles and neck stiffened. No thyroid enlargement and no cervical lymphadenopathy. Right pupil was dilated, with the right eye directed down. Left pupil contracted and in a central position. No exophthalmus. Extraocular movement could not be tested because of patient's lethargy. The ears were negative. The nose, throat and mouth were normal. Lung expansion free and equal but deep and slow. Tactile fremitus normal, percussion note normal and breath sounds normal. The apex beat was in the fifth interspace midclavicular line. No enlargement, murmur or displacement. Rate about 68. Abdomen negative; no muscular twitching or convulsions. Patient was stuporous and unable to cooperate at all. Did not respond to any stimuli. Knee jerks were hypertonic, the achilles reflexes present. There was bilateral ankle clonus, but the Babinski was absent on both sides, as were also the confirmatory tests. The reflexes of the upper extremities were present on both sides and there was no spasticity.

On admission, temperature 98.7, pulse 68, respiration 20. One hour after admission the patient was seen by Dr. T. H. Weisenberg, who made the following notes: "When I first saw the patient she responded to questions, giving the right answers, with the exception that she thought I was Dr. Coates. She was able to open her eyes and at that time her pupils were small but equally dilated. She was able to look up, down, but not to right or left. It was impossible for me to determine whether this was due to paralysis or her mental state, more probably the latter. There was some weakness in the lower

left face. Her history, obtained from the relatives, showed that she had recently complained of headache, which was progressively worse, and nausea and vomiting for the past two days. She has also been drowsy and somewhat lethargic, but there were no convulsive phenomena or ocular symptoms with the exception of some photophobia. On returning to the room after one-half hour, she was found much more stupid, the head drawn backward and to the right; could no longer answer questions; the left pupil was rigid and small; the right pupil dilated to its maximum and not responding to light. During the dictation of these notes, the right eye deviated downwards, with total lack of response to any type of stimulation. When first examined, power in right hand was normal and left hand diminished. At the second examination, the left foot was turned outward, the left hand in decerebrate position. Right knee jerk was increased; there was an abortive clonus; plantar irritation gave no response. Left knee jerk was more prompt than the right and clonus more persistent. Abdominal reflexes absent both sides. The evidence points to right frontal lobe abscess with even now a basal meningitis involvement. Fifteen minutes later there was present a bilateral Babinski. She used right arm freely but not the left. The right pupil had come down to same size as the left and the deviation of the right eye downward had disappeared. Throughout the examination she seemed to have a protrusion of the right eye."

She was then seen by Dr. Edmund M. Spaeth, who gave the following eye report: O. D., widely dilated and fixed; O. S., pin point and fixed. A short time before the pupil O. D. had been pin point as was now that of the left (after ten minute interval). Slight exophthalmos O. D., with widening of palpebral fissure. The eye was held closed. The right eye is in hypoduction. Fundus O. D. disc swollen and pale, margin blurred and edematous. The elevation of this nerve head is at least 2 diopters. There is no hemorrhage in the retina. O. S. nerve head seen also swollen and edematous with blurred margins. The elevation in this eye is from 1 to 1.2 diopters. In both eyes the veins of the retina are engorged and tortuous. There is no hemorrhage on the left.

The patient was taken immediately to the operating room and the following operation performed.

Operation, January 10th.—The usual Killian incision, but slightly beneath the eyebrow. After the periosteum was retracted a small hole was made into the sinus, which was apparently dry. The anterior wall of the sinus was now removed and the interior appeared to have regenerated, as far as inflammatory symptoms were concerned, except at the extreme upper apex. At this point, where the anterior and posterior sinus walls came together, a drop of pus was present. On probing it was found that the diploe appeared softened and somewhat purulent. A vertical incision was now made up the midforehead to gain necessary room, and after turning back the flap the external table over this area, although apparently healthy, was gradually removed in an upward direction until this osteomyelitic process of the medullary substance was completely uncovered and normal bone was encountered on all sides.

The internal table above and the posterior sinus wall below were sponged and carefully inspected in order to discover a possible focus of penetrating infection, but no discoloration or softening could be detected. Despite this negative finding, the extreme gravity of the patient's condition demanded instant action, so the internal table was opened above the upper limits of the sinus cavity and also immediately a few drops of heavy white pus followed the introduction of a probe between the dura and internal table. It was decided to lay bare a considerable portion of the frontal lobe, so the bone above the sinus cavity, as well as the posterior sinus wall was resected. The dura above began to bulge and did not pulsate while that portion directly behind the sinus cavity showed no bulging and pulsated faintly.

After a quick consultation, a scalpel was introduced into the bulging portion through the dura directly backward to the depth of one-half, three-quarters and finally one inch, which was followed only by a gush of dark blood. As the patient was now moribund and further operative interference could do no harm and might possibly be of benefit, the scalpel was pushed further inward and backward, when suddenly a gush of pus under great pressure welled out of the wound alongside of the scalpel blade, dividing into two streams as it flowed down over the patient's face and hair. The scalpel was quickly removed and a pair of forceps inserted, opened and removed,

thus opening widely the deep wound. Another gush of pus followed in even greater volume than the first.

With a long, thin bistoury a portion of the brain with the dura about the size of a small thimble was resected so as to give free access to the abscess. The abscess cavity was now packed with gauze, following which more purulent material was evacuated. The bleeding had now become so intense from the brain and meninges and from the abscess cavity that if allowed to continue it would soon have exsanguinated the patient. As all of the purulent secretion appeared to have been evacuated, the cavity was snugly packed with seamed iodoform gauze, one inch wide, oven ten feet being required to fill the cavity and sinus. The wound, was, of course, left open and the patient returned to her room in a precarious condition. After about seventy minutes, consciousness seemed to be trying to return, which surely was a most welcome manifestation, as she had been brought to the operating room to all intents and purposes moribund.

In the course of a few hours, complete consciousness returned, but she was still a mighty sick girl. She was put under morphia and atropin, and provision was made for blood transfusion at the earliest possible moment.

Postoperative Treatment and Course.—After operation, the patient, in the course of an hour, began to respond to suggestions. Both pupils were equal and about the same plane, but the eye signs quite variable during the next few hours. Blood pressure immediately after operation was 110/80, but two hours later was 90/40. A type 4 donor was located, cross agglutinated, and at 9:25 p. m. the patient was given 500 cc. of whole blood by the citrate method as well as 200 cc. of normal saline. The pulse remained of good quality and rate. At this time the patient was seen to move both arms and legs and, when asked to do so, put both arms under the bed clothing. Before the transfusion the temperature was 102 rectal, pulse 90, respiration 20. An hour later it was the same except the pulse had come down to 80. Patient was unable to answer questions. General blood examination immediately postoperative: Erythrocytes 3,850,000, leucocytes 17,450, hemoglobin 68 per cent, small lymph. 7, polymorph. 93. Urine showed a faint trace of albumen but was otherwise negative.

Culture from the brain abscess eventually showed a staphylococcus albus. Laboratory report from Dr. Eugene A. Case: "Examination of tissue removed at operation: The brain tissue shows a perivascular cellular infiltration of lymphocytes, plasma cells and mononuclears. The pia arachnoid is also infiltrated with similar cells. The sections do not show the lining of the abscess cavity."

Patient had a good night following operation, and the next day temperature, pulse and respiration remained about the same, with a recession of the temperature to 99.4 at its lowest point. Pulse full and regular, of good quality. Blood pressure 98/70. External dressings were changed and the patient's mental condition seemed clearer. She was given an enema and fluid by rectum and morphin sulphate for restlessness. Dr. Weisenburg reported patient much better, clearer mentally but still somewhat stupid. The left hemiplegia was about the same as previously, although the weakness of the face was slightly more marked. No further evidence of progressive meningitis. Under dilated pupils, Dr. Spaeth reported the eye condition unchanged. Blood examination was about the same as previously, except that the leucocytes had gone up to 24,550. The highest elevation of temperature for the 24 hours was 101.

On January 12th, her general condition was about the same, sleeping most of the time. White blood count had fallen to 17,740, blood pressure 110/70. Temperature ranged from 99 to 102 and pulse from 108 to 120. Patient's neck was still stiff; there was no nausea.

Fundus examination by Dr. Spaeth showed the papilledema unchanged but a definite decrease in the tortuosity and the engorgement of the retinal vessels. Patient conversed rationally with members of her family and did not complain of any discomfort or headache. On January 13, 1930, the temperature and pulse range were about the same as the previous day. There was some restlessness, taking fluids by mouth very well. The outer dressing was removed; 18 inches of iodoform gauze were removed from the wound. The edge of the wound was clean; no evidence of herniation. Dr. Weisenberg reported patient better mentally and physically from the neurologic viewpoint. Weakness of the left side of the face was less.

The Babinski was absent. Dr. Spaeth reported a definite bilateral paralysis of the external recti, slightly more marked on the left. Pupils reacting normally, but the left slightly smaller than the right. Papilledema about the same. Leucocytes, 14,350.

January 14, 1930. Patient slightly irrational at times. Leucocytes, 12,700. Wound was dressed and 18 inches of gauze removed, making a total of 36 inches. Neurologic signs improved. There was some mental upset in that the patient did not recognize people by sight, calling them by wrong names. Eye condition unchanged.

January 15, 1930. Temperature range between 100 and 101.4, pulse 92 to 110, respiration 20. Patient resting well; general condition very good. Neck still stiff; no headache. Thirty inches of gauze were removed from the wound, making a total of 66 inches. Patient still dull at times. This day all reflexes were absent with the exception of the achilles, which could only be obtained slightly. No further signs of meningeal involvement. At this time a very fine area of hemorrhagic extravasation was noted in O. S. between superior temporal artery and vein just as they enter the nerve head. Leucocytes, 17,550.

From now on the patient showed definite improvement day by day, the temperature and pulse gradually coming down and the stiff neck clearing up. Her mental condition shortly became clear, but sometimes the knee jerks were absent. The gauze packing of the wound was removed a little at a time daily. On the 17th, the hemorrhage of the left eye had entirely disappeared and there was definite improvement in both eyes. The hemiplegia had also practically disappeared.

On the 18th, 18 inches of gauze packing was removed from the abscess cavity itself, the previous removals having been only from the frontal sinus. By January 20, 1930, 8 feet 2 inches of packing had been removed from the sinus and abscess cavity. Dr. Spaeth reported diplopia field unmoved, diplopia constant in all fields, due to a convergent squint; paralysis of the external rectus more extensive on the left than on the right. Papilledema was beginning to recede in both eyes. Leucocyte count still showed an increase up to 16,000.

On the 22nd, twelve days after operation, all of the packing had been removed from the abscess cavity, which was cleansed and immediately repacked daily. General condition splendid, bright and cheerful, no headache or complaints, noticeable improvement in eye condition. Within ten days, although there was still some diplopia present, both eyes could be rotated externally to a degree almost normal. The abscess cavity continued to drain, but in the course of two or three weeks the frontal sinus cavity became largely obliterated by granulations, stiffness of the neck had entirely disappeared, and on January 27th pulsations in the frontal lobe of the brain were noticed.

On the 31st, papilledema had been reduced to 1 diopter in each eye. Leucocytes were 14,000 and temperature not over 99.8 with pulse 100 to 106.

A small sequestrum was removed from the wound February 6, 1930, at which time the patient was bothered by an erosion of an unerupted right third lower molar tooth, which caused some pain and temperature disturbance for a day or so, but which quickly subsided under treatment.

By February 10, 1930, reflexes in the legs were practically normal, though slightly stronger on the right side. General condition very much improved. Ocular movements still slightly retracted in upper deviation of the left eye. Pupils equal and react promptly and freely to light. In both eyes there was still some slight blurring present in the upper pole, somewhat less in the left.

After February 14, 1930, leucocyte counts were discontinued and the temperature remained practically normal. At this time the cavity of the abscess was estimated to be from $1\frac{1}{2}$ to 2 inches deep, packing being removed and replaced with fresh gauze daily.

After February 19, 1930, the abscess was gently syringed daily with bichloride of mercury 1/5,000, strengthened in the course of two weeks to 1/3,000. Constant improvement was noticed with the external wound healing nicely, with, of course, some deformity, and the eyes in all respects constantly improving. Drainage was less marked after the irrigations were started, but the daily packing of the abscess cavity with one-half inch iodoform gauze was continued.

She was kept in bed until March 10, 1930, after which time she gradually spent more and more time out of bed and was permitted to leave the hospital for short walks and drives after March 25th.

By April 7, 1930, the eyes were practically normal and the depth of the cavity of the brain reduced to about $\frac{1}{2}$ inch. Granulations at the lower part of the wound over the supra-orbital ridge continued to discharge a small amount of pus, and bare bone was felt with the probe. The remainder of the sinus cavity had by now been obliterated by firm, organized granulations.

On April 10, 1930, the remaining $\frac{1}{2}$ inch of packing was forced out of the abscess cavity, which promptly closed up. It was reopened with the probe, but as no fluid was found it was allowed to close permanently.

April 11, 1930. Two small sequestra were removed from under the granulations, and the sinus in the brain was observed to be definitely healed.

April 18, 1930. Two more very small sequestra were removed under a small area of granulation tissue down near the supraorbital ridge, about three-quarters of an inch below the site of the abscess opening.

April 19, 1930. Discharged from hospital. (This could have been done long before, but she resided at some distance from the city, which would have made it very inconvenient to come in daily for dressing.)

April 26, 1930. Wound entirely healed.

The following cases represent those which have been reported as recovering following operation:

Herzfeld.—*Berlin klin. Wochenschr.* November 25, S. 1180, 1901.

Male, 20 years. History of frontal sinusitis following cold: after six weeks became decidedly worse. Some temperature, pulse about 50, pain in head growing steadier in intensity; beginning stiffness of neck.

Operation.—Left frontal sinus opened; mucosa greatly swollen and discolored, and bulges upward through the wound and which on being opened contained only a small amount of fetid pus with granulations. Posterior sinus wall found carious, so removed and at lower part extremely fetid pus flowed

out from between the dura and bone, the source of which it was not possible to determine. The underlying dura was discolored and failed to pulsate, and on being incised gave forth a considerable quantity of very fetid pus. On retracting the dura a fistula extending into the brain was discovered. This fistula was dilated with the forceps, after which it was possible to put one's finger into an abscess hole up to the second phalanx, both laterally and superiorly. (Pus showed pneumococcus.)

The cavity was packed loosely with iodoform gauze and dressed externally with the same. The operation was immediately successful. Headache relieved entirely. Temperature became normal but pulse remained low. Profuse purulent secretion for six days, after which time it diminished, and healing quickly followed, so that the external skin wound at the end of five weeks became fully closed. One now could note only a slight pulsation, which became less and less until it practically disappeared. Patient entirely well with all faculties preserved. (No details concerning the dressings are given. A remarkable fact was that the eyegrounds at no time showed any changes.)

Rawling.—*Trans. Med. Soc. of London*, Vol. 30, p. 375, 1907.

Male, frontal headache with edema right upper eyelid.

May, 1905. Operation; roof of orbit exposed.

June. Complete coma and paralysis of left side of body and face. Second operation: Skull opened and frontal lobe exposed, abscess being found and evacuated.

July 15th. Discharged cured.

1906 (early). Another abscess in same situation, evacuated.

October 13, 1906. Headache severe; drowsy and irritable bulging over site of operation. Operation but no abscess found; marked hernia of brain which was shaved away.

October 24th. Abscess again sought and found. Abscess walls removed en masse. Recovery. Frontal sinus operated upon later. *Staphylococcus pyogenes aureus*.

(While it is stated that "another abscess was found," one cannot help thinking it was a refilling of the same abscess caused by too early discontinuance of drainage.)

Cargill, Turner and Thomson.—*Proc. Royal Soc. of Med.* (Laryng. Section), p. 125, 1907-08.

Male, 31 years. Left orbital cellulitis. Incised and pus evacuated. Two weeks later great depression; slow cerebra-
tion, irritable, temperature and pulse rate subnormal. Optic
neuritis marked. Left frontal sinus discharging pus, and bone
at bottom clearly carious.

Operation.—Frontal sinus opened; posterior wall softened
and easily broken down, exposing cerebral surface covered
with dirty gray looking granulations. No dura could be found.
The brain pulsated with no bulging. Exploratory needle
brought pus, 3 to 4 ounces being evacuated. Staphylococcus
aureus. The after-treatment was not described. Patient is
now quite well.

Donalies.—*Archiv. f. Ohrenhk.*, Bd. 75, S., 199, 1908.

Male, 12½ years. Brought into the office a very sick boy.
Pain in head, swollen nose and pus, some fever, pulse 98, and
answered questions slowly but correctly. There was a fluctu-
ating swelling in midforehead. History of traumatism four
weeks previous.

Diagnosis: Acute purulent bilateral frontal sinusitis; sub-
periosteal abscess; probable intracranial complications.

Operation.—Abscess opened by vertical incision and both
frontal sinuses opened; no improvement in general condition.

October 15th. Patient's head stiff and drawn backward;
pulse slow; both frontals discharging profusely.

October 15th. More apathetic, sleepy; condition worse.

October 21st.—Spasms of left side of body and face.

Operation.—Posterior wall of right frontal sinus removed.
Dura somewhat hyperemic but otherwise apparently normal.
Dura incised and frontal lobe punctured; no pus found. The left
frontal was now attacked and on opening the posterior wall
a few drops of thick pus was seen on the dura. The dura
itself was discolored but no break was found in continuity.
It was now incised through the discolored portion and at a
depth of 2 mm. an abscess was found about 1 to 1.5 cm. in
diameter. Immediate improvement followed the operation;
temperature lower and pulse higher. Uneventful recovery.
Wound closed November 8th. (No mention is made of man-
ner or method of postoperative treatment. The curious part
of this case was the spasms on the diseased side.)

Wiener.—*N. Y. Med. Record*, Oct. 22, p. 715, 1910.

Male, 28 years. Four years ago external operation for frontal sinusitis, which was followed by a secondary operation one year later. Since then recurrent attacks of excruciating headache radiating from right frontal sinus. Fistulous opening at inner third of brow discharging pus. Patient of late forgetful and irritable. Marked tenderness over right frontal sinus. Brain abscess suspected and operation advised but refused. Two weeks later became semistuporous, passing from one convulsion into another, these being brought about by the collateral edema.

Operation.—Right frontal sinus exposed and found to have been completely eradicated. On posterior wall a small black spot was noted, which admitted a probe to the depth of three and one-half inches. One-half dram of pus followed its removal. Incision enlarged and scalp elevated and area laid bare about size of one's hand. A spot in the dura about the size of a 25-cent piece was found discolored with granulations. A crucial incision was made in the dura into the abscess cavity. Pus permitted to flow out and cavity sponged until clean, which appeared to be about the size of a small child's fist, containing approximately four ounces of pus. Cavity wiped with 90 per cent alcohol and packed (not too firmly) with one-half inch selvaged iodoform gauze.

On the following day patient felt perfectly comfortable and outside dressings changed. On third day internal packing removed with little secretion in cavity. The cavity was permitted to fill up with granulations and was completely closed in seven weeks. Patient has all faculties and for the past year has had no recurrence of symptoms. He is actively engaged at his former occupation as a painter and appears to be perfectly well.

(This case appears to be the first one operated upon for brain abscess with recovery that had not undergone previous operations for other conditions. All of these cases with fistula formation seem to respond particularly well to surgical interference.)

Rische.—*Zeitschr. f. Ohrenheilk.*, Bd. 62, S. 231, 1911.

Male, 19 years. One month ago swelling noted over left eye which quickly spread to upper lid. Incised; abscess evacuated. Next day pain worse with vomiting. Temperature 39.

Operation.—Fistula into frontal sinus; anterior wall softened. Granulations in frontal sinus. Perforation in cerebral wall. Posterior wall resected. Dura covered with granulations and did not pulsate. Needle puncture into the frontal lobe brought pus at depth of 2 cm. A small knife was now introduced 2 cm. and about $1\frac{1}{2}$ to 2 tablespoonsful of pus evacuated. Rubber drainage tube in abscess cavity and iodoform gauze external in sinus.

September 8th (next day). Temperature fell to 37.6.

September 9th. Dressing soaked with pus; changed also new drain and tampon. Dressings changed daily.

September 17th. Rubber drainage tube discarded and strip of gauze substituted.

September 19th. Vomited during night and complained of headache and looked pale; pulse 58 and small. The dressings removed and opening in the dura dilated, whereupon several cm. of pus gushed out. A new rubber tube was again inserted. Unfavorable symptoms at once disappeared.

September 27th. Rubber drain again discarded.

October 10th. Both brain and dura entirely healed. External wound healed.

October 31st. Patient discharged.

(This case shows the danger of removing the means of drainage too soon.)

Berens.—*Laryngoscope*, p. 1083, 1916.

Male, 30 years. History of an old pansinusitis with external radical operation (January, 1913). Three years later frontal headache with large swelling of cicatrix in right frontal region.

Operation, January 5, 1916.—Old frontal wound reopened, with pus under slight pressure. Sequestrum on posterior wall, which on removal yielded about four drams of pus (external). Granulations were present, which were hiding a perforation through which a probe was passed in an upward and inward direction for a distance of $2\frac{1}{8}$ inches. This opening was gently enlarged with forceps and a horse hair inserted into the stem of the abscess. (Does not state if more pus was evacuated.) Wet gauze dressing. Free drainage followed. More strands of horse hair were added daily for a week until the fistula had become as large as a lead pencil. A soft rubber drainage tube was then substituted and the discharge became so profuse that

for two weeks it was necessary to change the dressings every six hours. Discharge practically ceased at end of five weeks but wound not allowed to heal until April 18th.

General symptoms after operation: Much frontal headache, restless and irritable, pupils contracted. Temperature 100 to 101. Culture, streptococcus hemolyticus pure.

Lynch.—*Trans. Am. Laryng. Assn.*, p. 144, 1917.

Female, married. More than 15 years suffered with headaches. Right maxillary sinus irrigated for a year with only varying relief. Middle turbinate removed, frontal opened intranasally. No relief. Leucocytes, 11,000. One year later (1913), external frontal. No improvement. In 1916, posterior ethmoid and sphenoid full of pus. Treatments relieved headaches but old symptoms soon returned. Posterior ethmoid cells and sphenoid opened, exposing cavernous sinus and dura. Progressed nicely for five days, then symptoms of meningitis which, however, after three days subsided. Four weeks later symptoms reappeared with 17,000 leucocytes. Mental dullness almost to coma, roused only with greatest difficulty.

Operation.—Old frontal wound opened, which contained about 15 drops of milky pus. Posterior sinus wall removed. Meninges appeared normal. Dura split. Needle introduced into frontal lobe, and at depth of 1 inch three drams of yellow-green pus withdrawn. After preparation of opening for a wire gauze drain the needle was reintroduced and four more drams of pus withdrawn. The brain was searched for abscess cavity but no evidence could be found. Finally a small rubber tissue drain was introduced into the brain to the depth from which the pus had been aspirated. Patient became conscious after recovery from anesthetic, and in six hours began to talk. Daily dressing reveals no pus and on sixth day drain removed and brain explored again for pus but without result. Wound kept open for two weeks, then allowed to heal. Recovery rapid; headaches gone; mentality clear and bright; leucocytes down to 5,800. Pus staphylococcus aureus in pure culture.

(This case on account of its chronicity undoubtedly had a marked abscess wall which collapsed where the purulent secretion was withdrawn. It would seem that while it may not always be necessary to lengthily drain these cases to obtain a cure, nevertheless the danger of secondary abscess formation

is so great that one takes a palpable chance in not so doing. To be on the safe side it is much better to allow the cavity to slowly fill up with granulation tissue, even though the duration of the hospitalization is considerably lengthened.)

Leegaard.—*ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, p. 108, 1919.

Male, 22 years. Sinus discharge for several months. Became worse after exposure from sleeping in a tent that was damp. Trouble centered around left eye. Radical operation on left frontal sinus. Discharged from hospital after six weeks, apparently well. Two weeks afterward was again admitted to hospital suffering great pain in left sinus coupled with dizziness. Old wound opened; no pus in sinus; posterior wall removed; dura bulging. Needle puncture brought thick pus $1\frac{1}{2}$ cm., 35 c. cm. evacuated slowly and carefully. Culture showed diplococci. Split drainage tube inserted 1 cm. Next day tube lay outside of dural opening. Strip of gauze inserted to keep opening patulous. Dressings changed daily; opening in dura separated each time with forceps.

This dural opening closed one month after operation. Exposed portion of dura began to bulge, and at the end of six weeks reached size of walnut. Gradually subsided and disappeared in about two months. Slight increases in evening temperature continued for three months, then ceased. Patient's general condition excellent; operation scar firm; slight diplopia which cleared up in about nine weeks.

(After the first day gauze packing was apparently used and drainage tube discarded. Hernia disappeared without special treatment.)

Kerr.—*Archiv. of Surgery*, Vol. 7, p. 297, 1923.

Male, 15 years. Two years before had a submucous resection, which was followed by cerebrospinal rhinorrhea. Two months ago rhinorrhea suddenly ceased and pneumococcus meningitis developed. Recovery followed spinal punctures.

After a period of time (not stated) headache with vomiting appeared with choked disc. Leucocytosis, 16,000. Neurologic examination negative. Symptoms pointed to infection in right frontal sinus region and diagnosis of frontal lobe abscess was made.

Operation.—Craniotomy was performed and several ounces of greenish pus (pneumococcus type IV) was evacuated from an abscess about 3 cm. deep in right frontal lobe. Drainage was maintained for six months followed by complete cure. (No details were given concerning technic of operation or after-treatment.)

Swindells and Rankin.—*British Medical Journal*, p. 622, Vol. 2, 1924.

October 27th. Male, 23 years. Edema of left upper and lower eyelids. Tenderness over left maxilla. Temperature 103, pulse 50.

November 7th. Headache increased.

November 9th. Operation: Left antrum drained through canine fossa. Left frontal sinus opened, which contained glairy inspissated pus. Patient greatly improved.

November 19th. Headache again severe. Patient became violent and abusive toward the nurses. Mind clouded; condition weak.

November 27th. Handgrips weak; right weaker; semi-comatose; pulse 48.

November 28th. Third operation: Skull trephined above and to left of frontal sinus. Dura bulged into opening with no pulsation. Marked granulating area. Dura incised and pus found at depth of three-fourths inch (micrococci and staphylococci), 5 ounces collected. A gauze drain, 2½ inches. Next day patient entirely changed; mind clear and calm. Twelve days after two epileptiform seizures and similar attacks.

January 6th. All packing removed.

January 20th. Wound allowed to close.

February 6th. Discharged cured.

(Five ounces of pus is perhaps the largest frontal lobe abscess reported.)

Wishart.—*Journ. of Laryng.*, p. 638, 1924.

Female, 10 years. Operated upon for chronic frontal sinusitis. Abscess found while probing through defect in posterior wall. Half ounce of pus evacuated. (Staphylococcus albus.) Drained with rubber tube. Uninterrupted recovery.

Jeseman, Boston.—*Med. and Surg. Journ.*, Vol. 192, p. 739, 1925.

Male, 39 years. Right frontal sinusitis.

Operation, February 23rd.—Intranasal, anterior end middle turbinate and anterior ethmoid cells opened.

February 25th. Convulsion which lasted 10 minutes. Semi-conscious. Diagnosis, brain abscess.

Operation, February 25th.—Frontal sinus opened and filled with thick dark colored pus. Entire membrane pyogenic. Entire front wall removed; no defect found in posterior wall. Large opening made into this wall. Dura darkish and appeared under tension without pulsating. Incision through dura brought no pus but at depth of one-fourth inch an abscess was opened which contained a large amount of dark colored pus. Two rubber tubes used for drainage. Wound allowed to remain open.

Following days, pain improved, drowsiness less marked and mind clear. Profuse discharge. Temperature 101 by axilla. Rubber drains not changed for one week, then only one returned.

March 20th. Drains omitted.

April 15th. Plastic closure of wound with healing by first intention.

April 23rd. Dismissed from hospital.

(It is most unusual to find an abscess at such a superficial depth; the drainage difficulties in such a case would be markedly lessened.)

Arnoldson and Bostrom.—*Acta Oto-Laryngologica*, Vol. 8, p. 346, 1925.

Male, 14 years. May 15th, admitted to hospital for acute frontal sinusitis; under treatment for sinusitis since May 15th.

May 24th. Intranasal removal of middle turbinate and passage into frontal sinus enlarged; no pus.

May 29th. Frontal sinus opened externally. Contained pus and granulation tissue. Posterior wall removed; dura discolored and tense. Puncture above brought fetid pus (sterile). Opening in dura enlarged and an abscess uncovered the size of an egg. Drain introduced. Tampon of iodoform gauze. Cure, discharged June 22nd.

(Authors do not state manner of dressings nor postoperative progress of patient. It will be noted that two weeks in the hospital elapsed before the presence of the brain abscess was recognized and operation performed.)

Boss.—*Zeitsch. f. Laryng. u. Rhinol.*, Bd. XIII, S. 128, 1925.

Tendency to secondary abscess formation is well shown in Boss' case 1, which required five incisions.

Original operation (puncture of frontal lobe to depth of 3 cm.) November 4th. The second followed in 2½ months, January 19th; the third in a little over three months, April 22nd; the fourth in nine days, May 8th, and the fifth about six months, November 5th. Patient died in two days. Autopsy showed another abscess in frontal lobe which had not been opened.

To my mind, all this was due to the fact that drainage had not been kept up, and after each operation the patient was permitted to leave the hospital too soon. His second case went on to a more favorable conclusion.

Case 2.—Male, five days after an automobile accident complained of pain in right frontal sinus with edema of right eyelid.

August 14th. Local treatment for acute frontal sinusitis.

August 18th. Headache worse; middle turbinate removed. Improvement in headache and edema.

August 30th. Symptoms return, even more marked. Killian radical on frontal sinus. Sinus filled with gray-green pus.

September 4th. Condition much better; wound closed.

September 6th. Headache reappeared and temperature 38.5.

September 11th. Wound reopened, considerable pus appeared at inner angle. Headache becoming unbearable. Temperature 38.8. (During following nine days patient's condition gradually worse.)

September 20. Continued vomiting; headache and beginning somnolence. Neurologic examination, beginning right facial paralysis.

Operation.—Removal of apparently intact posterior sinus wall. Dura showed bluish-red discoloration about the size of a quarter. Pulsation present. Puncture 1 cm. deep brought one ounce gray-green pus.

September 24th. Dilaton of passage to abscess cavity; tamponade.

September 25th. Dressing changed daily. Irrigation with rivanol, 1/5,000. Uninterrupted healing.

(This is a typical case of frontal sinus infection with frontal lobe abscess formation where the latter remained unrecognized until it began to affect certain vital centers. Continued headache after a radical operation is, other things being eliminated, pathognomonic for meningeal involvement. The longer it continues without stiffness of the neck, the more indicative of some form of brain abscess.)

Callfas.—*Trans. Amer. Laryng., Rhin. and Otol. Soc.*, p. 436, case 2, 1926.

Female, 28 years. History of cold after swimming. Much pain and swelling in and around left orbit. Patient semi-conscious.

August 8th.—Operation: Incision below supraorbital arch and rubber drainage tube inserted. Next day both ethmoids exenterated, followed by some improvement. Blood positive for staphylococcus. Next three months variable with abscesses in breast and thigh.

November 24th. Patient much worse. Gave very slow response to questions. Neurologic findings suggest a lesion in left hemisphere.

Operation, November 25th.—Left frontal sinus opened; posterior wall badly diseased. Needle puncture brought thick yellow pus at depth of 3 cm. (staphylococcus aureus). Next day patient showed improvement, which continued until she left the hospital. (Reporter does not mention kind of dressings or treatment except intravenous injections of 1 per cent solution of mercurochrome, which seemed to do more harm than good. Perforated rubber tubing may have been used for drainage. Callfas uses a rubber tube, splitting the protruding end into four segments which are folded back and stitched to skin. The tube is gradually shortened as indicated. This case is typical of the manner in which some of these frontal lobe abscesses gradually progress.)

Mathieu and Peron.—*Paris Medical*, October 2, p. 270, 1926.

Case 2.—Male, 22 years, syphilitic.

Operation.—Cure.

Portman.—*Bull. et Mem. Soc. Med. et Chirurg. de Bordeaux*, p. 365, 1926. Males, 28 years.

Operation.—Cure.

Winter and Kalt.—*Rev. d'oto.-neuro.-ocul.*, p. 702, 1926.

Operation.—Cure.

Alvin.—*Lyon Medical*, T. 140, p. 127, 1927.

Female, 23 years. During a voyage to South Africa contracted a heavy cold. Disembarked at Tunis and was operated upon for a collection of pus above left eye. Arrived at Marseilles April 19th. Shortly afterwards vomiting set in and loss of memory. Pulse below 60. Reflexes diminished.

Operation, May 1st.—Frontal sinus opened; two fistulas found on floor, one surrounded by granulations. Resection of the inferior wall of the sinus with drainage into the nose. (Author gives no further data regarding operation or dressings.) One hour after operation pulse returned to 80. Lumbar puncture shows no sign of meningitis. At the end of 36 hours pulse again falls to 60, and the torpor which had diminished reappears. Temperature 38.

Second Operation, May 9th.—Resection of part of the anterior sinus wall, and one finds a fistula at the top of the internal wall which had been overlooked at the first intervention. Resection of the internal wall showed meninges and dura apparently normal. (No further operating was done.) During the afternoon the patient's condition became much worse; pulse below 55, with incontinence. Semicomatose.

Third Operation, May 10th.—No anesthetic required. Dura incised; frontal lobe punctured, using a large trocar with immediate evacuation of an abscess containing about a large glass full of serous pus (*streptococcus*). Immediately after the operation she seemed to return to life. While she had not spoken for 24 hours, she responded to questions and her psychological state seemed completely changed. Pulse revived to 80. (No description of technic or drainage is given.) During the following days the suppuration was profuse. Four days after operation erysipelas appeared on face. Antistreptococcic serum administered. The temperature fell rapidly and at the same time the suppuration diminished.

May 31st. Drain removed.

June 26th. Central hernia the size of the palm of one's hand.

October 30th. Discharged.

(It is unfortunate that the author does not go into details regarding the technic of finishing the operation, as well as the

dressings and subsequent treatment. Such information is of inestimable value to those having a similar case under their care.)

Mackenzie Brown.—*ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY*, p. 710, 1927.

Male, 19 years. After swimming developed pain and tenderness over left frontal sinus. Temperature 103. Four days later swelling appeared; opened; pus cultures, streptococci. Well for four days; severe headache; ptosis of left eyelid. Patient vomited several times during the day. Became unconscious.

Sinus opened through old incision. Posterior wall removed, followed by about 1 dram of pus (extradural). Dura thickened and covered with granulations. No perforation found. Drain placed in sinus and wound closed. Improvement followed but on the sixth day after the operation he complained of headache and soon became unconscious. Old wound reopened. Dura opened and one-half ounce of pus was found one-half inch from the surface (streptococci). Drain inserted into abscess cavity. (Kind not stated.) Consciousness returned next day and no headache. Complete recovery followed. (Author gives no details regarding dressing or length of stay in hospital.)

Imperatorii.—*Trans. Am. Laryng. Assn.*, p. 34, 1927.

Reported five cases of frontal lobe abscess following frontal sinusitis. All of these underwent operation with two complete cures.

Case 1.—Male, 27 years. Headache in frontal region for seven weeks. Swelling over left frontal; incised; considerable pus evacuated. Headache persisted.

Radical Operation.—Inner table of both frontal sinuses necrosed with epidural abscess. Sinus found which led into right frontal lobe. Abscess evacuated containing about one ounce of pus. No culture reported. Drain placed in frontal lobe and wound left wide open. Headache disappeared within a week. (The writer does not go into details regarding the postoperative treatment.)

Case 2.—Female, 14 years. Headache for three weeks with increasing severity and periods of violent delirium. Temperature 103 to 105. Fluctuating mass over frontal region.

Radical Operation.—Both frontals contained pus. Inner walls apparently not involved. No improvement. Four days later wound again opened. Portion of posterior wall removed. Dura congested; no pulsation. Search into left frontal lobe proved negative. Small epidural abscess above frontal sinus on right which covered a sinus leading into the frontal lobe. Evacuation of two ounces of pus. No cultures reported. Pulsation immediately returned. (No detail of postoperative dressings. Patient left hospital in ten days. Author states he is still living.)

Sandes and Halloran.—*Medical Journal of Australia*, p. 298, August 27, 1927.

January 25, 1927. Male, 21 years. Headache and vomiting for three months following a head cold; cerebation slow; double papilledema.

January 26, 1927. Patient became comatose; pulse 130.

Operation.—Trephine in right frontal bone midway between eyebrow and hair of scalp. Exploratory needle brought pus. Needle track dilated and glove drain inserted. Eight ounces of pus were slowly evacuated. *Staphylococcus aureus*.

February 1st. Clear mentally and condition greatly improved. Much fluid was escaping and portion of abscess wall extruding.

March 30th. Small piece of granulating cerebral tissue removed from center of wound, revealing a cavity, the probe sinking in two inches with drain inserted.

April 21st. Patient discharged with no evidence of hernia and healed scar. He was practically blind in right eye from secondary optic atrophy.

(The enormous amount of pus evacuated makes this case noteworthy. We have been unable to find another case of frontal lobe abscess where more than five ounces were found, and this amount in only one instance.)

Motley.—*Southern Med. and Surg.*, p. 243, 1927.

Male, 10 years. Acute exacerbation of chronic purulent left pansinusitis with orbital cellulitis.

Operation, September 9th.—Radical frontal, ethmoid and sphenoid (external) on right side. Dura not exposed.

September 30th. Discharged. Condition good.

October 18th.—Slight swelling and purulent discharge through scar. X-ray showed area of osteomyelitis.

Operation.—Sequestrum about 2 cm. in diameter removed, which involved only outer table.

October 30th. Nausea and vomiting. Discs show $2\frac{1}{2}$ diopeters; choking of disc.

Operation.—Dura exposed $2 \times 1\frac{1}{2}$ inches and painted with iodine after waiting forty-eight hours for walling off; needle introduced and pus found at depth of 1 cm. Crucial incision through dura and Mosher drain placed inside of abscess; one ounce thick creamy pus evacuated (staphylococcus). Iodoform gauze packing. Dressings changed twice daily. Prompt relief to both nausea and vomiting. Wire basket removed from abscess two weeks after operation.

December 15th. Fourteen weeks later. Patient up and about. Small brain hernia, 3 cm. in diameter, which receded rapidly after spinal punctures and magnesium sulphate dehydration. Discharged. Remained well for past six months when last seen.

(This case shows the possibility of the abscess being multiple and cure following its recognition and proper treatment, even though delayed over many months.)

Sprowl.—ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY, p. 922, 1928.

Case 1.—Male, 40 years. Four months previously had developed a head cold with pain and tenderness over right eye which has persisted. Two months later vomiting began. The headaches increased in severity with impairment of vision and vertigo. Frontal sinus tender; thick greenish purulent discharge from right nose. Fundus revealed a bilateral papilledema. Temperature 101.4, pulse 56. White cells, 16,000.

Operation, April 8th.—Frontal sinus full of thick greenish pus under pressure. A large portion of the posterior wall was necrotic and lying free in the sinus. Through this perforation, which was about the size of a dime, the dura was seen covered with perforations which surrounded a fistula from which pus was exuding. The fistula was enlarged and three ounces of pus evacuated from the frontal lobe (streptococcus hemolyticus). The needle entered to a depth of three inches without reaching bottom. A rubber tube was introduced for drainage

and sutured to the skin flap. Iodoform packing around the tube. Wound dressed daily; convalescence uneventful. The tube was gradually shortened and entirely removed on May 26th (48 days). Wound entirely healed on June 4th. Patient last seen over two years later with no recurrence of symptoms.

Case 2.—Male, 14 years. On August 8th admitted to hospital with marked swelling and tenderness of right upper eyelid and frontal sinus. Edema of bulbar conjunctiva; pupils equal; fundi negative. Temperature 104.2; pulse 124. Leucocytes, 18,700.

Operation, August 10th.—Brow incision with evacuation of large orbital abscess; cigarette drain and wound left open. Patient improved daily and although still a slight discharge from wound was discharged on September 3rd.

On October 13th, wound still discharging. Skiagraph shows unusual shadow over right orbit, presumably due to bone destruction.

October 20th. Headache again appeared. Tenderness over frontal bone; temperature 98; pulse 84. Neurologic examination negative. Right disc beginning optic neuritis. Leucocyte, 16,400.

Operation, October 21st.—In outer angle of frontal sinus both plates necrotic. Small perforation in posterior plate with granulations on underlying dura. Osteomyelitis in outer plate of sinus extending above. On enlarging perforation the dura bulged upward into the wound. Incision one-half inch long in dura. Three puncture wounds in different directions were made into the frontal lobe but no pus appeared. The dural incision was packed with iodoform gauze and the external wound left open. Dressings changed daily, being soiled with cerebrospinal fluid. On third day a small hernia appeared through opening in dura which increased in size. During the next fourteen days patient gradually became worse, and on the fifteenth day severe headache and nausea appeared and the papilledema increased to one and one-half millimeters. Babinski positive.

November 6th, Operation.—Old incision enlarged and considerable softened bone removed. Dural wound enlarged and a brain searcher introduced just above the hernia and at the

depth of one inch an abscess found. The cavity was unroofed, as described by King, and one-half ounce of pus evacuated. The wound was dressed daily by covering the hernia with rubber tissue coated with vaselin and gauze over this. (No mention is made of the abscess cavity itself.) On the third day the hernia became quite large but slowly subsided after spinal puncture.

January 5th. The wound was entirely healed and the patient left the hospital. He remained free from symptoms until January 25th, when he returned complaining of severe pain around the eyes and appeared ill. The wound was bulging and the brain seemed tense. Leucocytes, 20,400. Kernig positive. The following day was distinctly worse.

January 27th. Old incision again opened. Brain searcher directed backward. Located a large superficial abscess containing about one and a half ounces of thick green pus (pneumococcus). This abscess was also unroofed and the brain permitted to herniate.

Convalescence was prolonged but uneventful. The large hernia was gradually reduced with spinal taps.

March 19th. Wound completely healed and discharged from hospital one week later.

(This case is particularly illuminating, for it shows, in the first place, the possibility of overlooking a frontal lobe abscess. Secondly, the tendency to the formation of secondary abscess, even under skillful management, and thirdly, the gradual disappearance of the hernia following spinal tapping. No less remarkable is the resistance shown by the younger patients after the purulent collection has thoroughly been evacuated.)

Learmonth.—*Surg. Clinics of N. A.*, August, p. 979, 1929.

Male, 6 years. Acute right frontal sinusitis with fistula over right supraorbital area.

Operation, October 17, 1927.—Wound broke down and drained until January. Examination of nose and sinuses negative. X-rays negative.

Diagnosis: Osteomyelitis of low virulence. Treatment: Ultraviolet ray. Sinus healed after one year. (February, 1928.)

July. Swelling reappeared over frontal sinus with headaches. Incision healed and relieved all symptoms.

September 24th. Sudden attacks of headache with projectile vomiting. Headache only relieved by lumbar puncture. Examination showed choked discs 3 to 4 diopters; vision greatly impaired, 6/30. Leucocytes, 16,300. Diagnosis: Abscess of frontal lobe.

Operation, October 29th.—Both frontal sinuses chronically inflamed; posterior bony wall appeared perfectly normal. This wall removed; dura apparently healthy. A blunt canula was passed into the frontal lobe and encountered resistance at depth of 4.5 cm. A needle was introduced and 15 cc. of thick grayish pus withdrawn by a syringe (staphylococcus). A tract was then made into the abscess and two drainage tubes placed in position and sutured. Iodoform gauze. Patient not much improved.

Operation, November 8th.—Passed needles in different directions, and on the lateral aspect of the frontal lobe encountered a second abscess from which 20 cc. pus was evacuated. Opening made into this abscess through the temple. The wall between the two abscesses broken down and a rubber tube passed from the frontal opening to the temporal opening. Postoperative course uneventful.

November 17th. Patient left hospital.

December 18th. Tube removed and wound kept open with gauze soaked in 1/1,000 acroflavin.

January 4th. Patient dismissed entirely cured.

(This case shows the importance of searching for other collections of pus when the symptoms do not show improvement. Many fatal cases reported in which this was not done give a similar history to this one before the secondary operation.)

Mayer.—*Wiener Med. Wochenschr.*, Bd. 79, S. 253, 1929.

Male, 20 years. Operation—Recovery.

J. E. J. King.—Personal communication, 1930.

Male, 19 years. Six weeks ago had a severe head cold, followed by left frontal sinusitis, which was operated upon. Since has had severe headaches. Vomits several times daily, which is projectile, and not preceded by nausea. No fever, pulse 58. Vision good, although fundus shows slight choking of disc with punctate hemorrhages. Appears to be mentally sluggish. Diagnosis: Brain abscess over right frontal lobe.

Operation, November 9th. — Trephine opening over right frontal lobe. By means of an exploratory puncture with a brain cannula into the right frontal lobe the typical resistance of a brain abscess was encountered at a depth of 3 cm. After the abscess had been located, a circular opening in the bone about $1\frac{1}{2}$ inch in diameter was made directly over the extreme end of the frontal lobe and the dura incised in a stellate fashion. The frontal lobe was then incised, and after removing a portion of the extreme tip a good exposure of the yet unopened abscess cavity was obtained. Before opening the abscess small iodoform gauze strips were introduced beneath the dura to prevent spreading of infection to meninges. An incision was now made into the abscess cavity, and thick, yellow, odorless pus removed by sponging and washing out with Dakin's solution. A section of the abscess wall which had a firm capsule was excised but not as much as was desired in order to allow complete herniation. Gauze dressing wet with Dakin's solution and rubber dam over abscess. Homely instillation of Dakin's solution.

Patient's condition showed immediate improvement; pulse up to 90 to 96.

November 11th. First dressing: Operative area clean. Dressing removed except around site of defect and hernia. Patient alert; talks coherently; no paralysis; temperature 99.6; pulse 78.

November 12th. Second dressing: Hernia projects 1 inch above skull. Dakinization every hour continued. Patient much improved. Soft diet.

November 14th. All original gauze removed and partially replaced. Dakinization.

November 22nd. Abscess cavity completely obliterated.

January 12th. Hernia found completely collapsed.

February 2nd. Sinus closed; patient discharged completely cured.

(The author goes into minute detail regarding the daily appearance of the wound as well as the dressings and the exact medication. Particular attention is paid to the hernia and the manner and method of treating same.)

In reviewing the treatment of brain abscess, at least as far as the frontal lobe is concerned, it seems to us that great stress is laid upon certain procedures which appear to be quite unnecessary, while other measures of the utmost importance are only given passing mention or for the most part entirely ignored. It is our purpose to present our experiences concerning the former and to enter into some detail regarding the latter, to the extent that their importance would seem to indicate.

In order to cure a brain abscess, we must first recognize its presence. Eagleton stated, in 1922, that at least 45 per cent were only diagnosed on the autopsy table. Since that time some improvement has occurred, but it is only within the past two years that expert neurologic examinations have played a great role in arriving at definite preoperative knowledge. So that one of the prime requisites for an ultimate cure lies in the early recognition of the pathologic condition. After the diagnosis of a frontal lobe abscess is definitely established, what procedure shall be adopted? If we study in detail those cases reported in which a fistula has formed we are at once impressed by the fact that most of these recovered.

Now, in considering the operative interference, why not attempt to emulate Nature as much as possible by making a fistula that is of sufficient size for adequate drainage and by packing to keep it patulous until the abscess cavity is closed by the slowly forming granulation tissue? This briefly is the method followed in our case just reported. This will necessitate approaching the meninges through the diseased frontal sinus—in other words, through a septic area.

We have found that a clean field is absolutely, in such cases, unnecessary; therefore, packing off the intradural space around the operative field is probably a waste of time. It was previously thought that the high mortality was due to exposing the meninges to purulent secretion with resulting meningitis. This has not proven to be the fact, for if the patient survives the operation the cause of death is usually a secondary abscess, at least as far as the frontal lobe is concerned. Suppose the approach had been made through the frontal sinus, the posterior wall of which has been removed and the dura exposed. What is the next step? To find, of course, the abscess. We

believe the frontal lobe should be penetrated with a bistoury until the purulent secretion is located. Penetration can be made to a depth of $2\frac{1}{2}$ cm. without danger to the lateral ventricles, and if the knife blade is not higher than $1\frac{1}{2}$ cm. from the floor the penetration can go as far as 4 to 5 cm. When pus appears a grooved director may be introduced; then a hole made directly into the abscess cavity the size of a lead pencil by cutting away that much dura and brain tissue. This opening should be large enough for adequate drainage, but too small for brain herniation, which is one of the banes of post-operative treatment. The abscess cavity can now be completely evacuated by sponging with gauze strips, although severe hemorrhage will probably be encountered, which, however, is controlled by the next step. The cavity is washed out with 1/3,000 bichloride solution, then packed snugly but not too tightly, with one-inch seamed iodoform gauze. The main purpose of this is to not only control the hemorrhage but to prevent collapse and adhesion of the abscess cavity walls, which would cause the retention of pus in the recesses and ultimately lead to the formation of a secondary abscess. There is, of course, the question as between the use of gauze packing and rubber drainage tubes, but we believe if a straight drainage tract into the abscess cavity is procured that the gauze will suffice, as we can then control the caliber of the tract by the pressure of the gauze.

The first intracranial dressing should be left ten days or two weeks and the gauze then removed piecemeal, but the outside layers of gauze can be changed once or even twice daily, depending upon the secretion, which usually is very profuse. This, to our mind, is the crux of the entire situation—that is, the drainage, which should be ample, continuous and particularly lengthy, for most of the failures have resulted from too early discontinuation of this essential part of the treatment.

The iodoform gauze is slowly removed on succeeding days, taking only that which has become loose and does not adhere to the brain substance. As the tract becomes shallowed the iodoform gauze is entirely removed at each dressing and replaced with fresh. In the meantime the patient should be confined to bed for at least five or six weeks. Finally, the tract will close and then will come the plastic work of reconstruction.

CONCLUSIONS.

1. All cases of frontal sinusitis about to undergo an external operation, that show any symptom pointing to the possibility of intracranial involvement (unusual headache, fundus changes and particularly some disturbances in the reflexes) should have a thorough neurologic examination.

2. The approach to the frontal lobe abscess should be through and above the diseased frontal sinus.

3. Abscess when found should have straight drainage about the size of a lead pencil. This will not only suffice but prevent a hernia forming. (If no capsule has formed on account of being in the acute stage a larger opening may be necessary.)

4. The drainage should be ample, continuous and lengthy until granulations fill the cavity.

It is our belief that close application to these principles will greatly reduce the mortality of frontal lobe abscess, at least in those following infection of the frontal sinus.

BIBLIOGRAPHY.

1. Denker: (Male, 17 years.) *Archiv. f. Laryng.*, Bd. 10, S. 411, 1900.
2. Discussion to Berry: Brain Abscess of Para-Nasal Origin. *Trans. Am. Laryng. Assn.*, p. 194, 1923.
3. Eagleton: Brain Abscess. *McMillan*, p. 251, 1922.
4. King: Treatment of Brain Abscess. *Surgery, Gyn. and Obs.*, p. 554, November, 1924.
5. Eagleton: Intradural Complications of Aural and Nasal Origin. *Arch. of Otolaryngology*, p. 60, July, 1925.



Fig. 1.

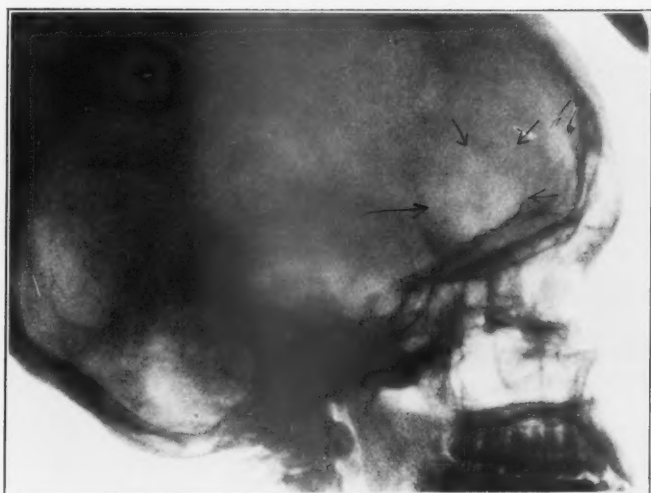


Fig. 2.

XXVIII.

THYMUS GLAND HYPERTROPHY AND ITS SIGNIFICANCE TO THE OTOLARYNGOLOGIST.*

A REPORT BASED ON X-RAY EXAMINATION OF TWO HUNDRED AND ONE CHILDREN, PREVIOUS TO TONSILLECTOMY, AND A REVIEW OF THE LITERATURE.

By JAMES A. FISHER, M. D.,

ASBURY PARK, N. J.

"The first mention of the thymus gland in literature was made by Galen, Rufus and Polydenker. However, it was not until the seventeenth century that anatomists described it, Vesalius in 1650 and Bartholinus in 1653."¹

In early medical history it was believed that the gland had excretory ducts emptying into one of the large thoracic veins, esophagus, trachea, pericardium or pleural cavity. During the eighteenth century some authors were of the opinion that the thymus gland should be regarded as a lymphoid organ, but it was not until the middle of the nineteenth century that the nature of the gland became real controversy, some contending that it was an epithelial organ similar to the thyroid, adrenals, etc.; others, that it was lymphoid in character. During the last half of the nineteenth century the anatomy and histology of the thymus became much better known.

Hammar states that the thymus may be divided into three types, cervical, thoracic and cervicothoracic. The hog and mice possess the cervical type, man the thoracic type and sheep the cervicothoracic type.

EMBRYOLOGY.

The thymus takes its origin by proliferation of the epithelium of the third branchial cleft, thus becoming entirely an endodermal organ. When fully developed the thymus may be

*Thesis to be presented before the Otolological, Rhinological and Laryngological Society.

regarded as composed of two pyramids, the right and left, the bases of which lie on the pericardium and whose apices extend up to the cervical region.

Besides the formation of the thymus from the third branchial cleft there is a parathymus formed from the fourth branchial cleft. This never reaches any very great degree of development. It gives rise to the accessory thymic nodules.

GROSS ANATOMY.

The thymus is composed of two distinct lobes covered with the thymic capsule which extends inwardly, sending off septa which divides the gland into lobes. It is rosy color in the fetus, grayish white in the newborn and rather yellowish later in life.

It has a fine lobulated surface, lies posterior to the manubrium and corpus sterni down to the level of the third or fourth intercostal cartilage, sometimes to the seventh or eighth.

Posteriorly the thymus lies upon the right auricle, is intimately attached to the left innominate vein and comes only in loose contact with the right. On the left side the thymus is in intimate contact with the vagus and inferior laryngeal nerves. Furthermore, the gland lies upon the cardiac plexus. It derives its blood supply from the small vessels taking their origin from the superior and inferior thyroid, internal mammary, innominate, intercostal and pericardicophrenic arteries.

"The existence of lymphatic vessels within the thymus was proven by Severance by careful injections. Hammar² was also able to ascertain the existence of such vessels."¹

Crotti says: "We may say that the thymus, like the thyroid, possesses a closed lymphatic system into which the organ empties its secretions and which finally reaches the blood circulation."

The nerve supply seems to be entirely sympathetic, no proof having been produced that the vagus supplies the gland.

HISTOLOGY.

The thymus is formed of lobules varying in size from 4 to 11 mm. Ordinarily they are of long oval shape and usually

separated from one another by thin layers of connective tissue. It is divided into two parts, the cortical and medullary.

The cortical portion is composed of a large amount of small dark cells with very little protoplasm, resembling histologically the genuine small lymphocytes. These are known as thymic lymphocytes. Besides these cells, neutrophiles, eosinophiles, plasma and mast cells are present in the cortical portion.

The medullary substance shows the same elements found in the cortical substance with the exception that plasma and mast cells are absent and lymphocytes are less numerous than in the cortical portion. Here are also found Hassal's corpuscles.

Crotti¹ says that "the thymus gland is an epithelial organ invaded by lymphocytes, and that these cells proliferate within the gland and become later the bulk of the thymic tissue. The epithelial component, however, persists as the reticulum and Hassal's corpuscles. The framework of the gland in the meshes of which lie the thickly crowded lymphocytes, is formed by the reticular epithelial cells. Thus the thymus is a lympho-epithelial organ."

INVOLUTION.

It was formerly thought that the thymus grew up to the second year after birth and then gradually diminished in weight and disappeared more or less entirely after the thirteenth year of life. But the accepted opinions now are, according to Crotti, that the thymus increases in weight up to puberty and during this time reaches its development, then undergoes physiologic involution but never disappears entirely during life.

According to Hammar,² involution may be divided into five stages: In the first, or infantile stage, connective tissue formation is only moderate and the parenchyma is still abundant. The second, or juvenile stage, in which the connective tissue formation is far more abundant, but in which the parenchyma remains about the same. The third, the young adult stage, in which the connective tissue is still more abundant and the reduction of the parenchyma into the cortical substance is al-

ready quite marked. The fourth, the adult type, in which the connective tissue only is abundant and the parenchyma much diminished, but fatty degeneration already taking place. Fifth, the senile type, consisting mostly of connective tissue containing islands of parenchyma cells. Mytotic figures can still be found, proliferation of Hassal's corpuscles still taking place, thus showing that the thymus is still a functioning organ.

Dr. Edith Boyd³ found the average weight of the thymus in well nourished children to be 13 gms. at birth, 20 gms. at six months and 35 gms. at thirteen years. The fluctuation in weight followed the fluctuations in body weight. Paltauf⁴ called any thymus over 15 gms. in weight excessive. Leonard Williams,⁵ in 1922, stated that its size changed with the change in climate and altered with the seasons, being larger in spring and summer.

From the study of the chemistry of the thymus it would seem that one of the most important functions is the metabolism of phosphorus, which may be compared to the metabolism of iodine in the thyroid.

That the thymus gland is not essential to life was proven by Park and McClure,⁶ in 1919, after experimental work conducted on dogs. They concluded that the extirpation of the thymus produces no detectable change in the hair, teeth, contour of the body, muscular development, strength, activity or intelligence of the animals. Also that it probably produces no alterations in the organs of internal secretion, but that the thymus probably produces a hormone. However, this has not been proven to date. The real purpose of the thymus has not been solved.

STATUS THYMOLYMPHATICUS.

Thymus hyperplasia is frequently combined with hyperplasia of the entire lymphatic system. In status lymphaticus the tonsils and adenoids are enlarged, the lingual tonsil, spleen and lymphatic follicles of the alimentary tract generally are enlarged. When the above is combined with thymus enlargement it is known as status thymolympathicus; when the thymus alone is enlarged, as status thymicus.

THYMIC DEATH.

What pathologic or physiologic act takes place in so-called thymic death is still a problem to be solved, although many theories have been advanced. Plater,⁷ in 1614, according to Ruhräh's "Pediatrics of the Past," was the first to describe a death from so-called thymic compression of the trachea.

Thymic tracheostenosis produces dyspnea ranging from labored respiration to violent choking spells, constant or intermittent, with or without acute paroxysms which either result in death of the child or gradual return to normal. This condition is usually found in the first few weeks of life, and the incidence diminishes during the second year of life.

Pressure in thymic tracheostenosis takes place at either the superior opening of the thorax or in the thorax. The thymus follows the up and down movements of the larynx and trachea during the various acts of swallowing, coughing or hyperextension of the head. It is then that the superior poles of the thymus become wedged behind the manubrium sterni and constrict the trachea in the so-called "critical space of Grawitz." This phenomena does not explain those sudden deaths which have shown none of these symptoms. Those occurring at induction of anesthesia, cold plunges, etc., are possibly explained by sudden swelling of the thymus pressing on the cardiac plexus.

Symmers⁸ states that sudden death in status lymphaticus may be brought about in two ways: "First, it may be in the nature of an anaphylactic reaction due to sensitization of the body by a specific nucleoprotein formed in the lymph nodes as a result of necrosis of numerous germinal follicles. Before the so-called anaphylactic incubation period has expired, the tissues are again subjected to the action of the same protein formed in the same type of tissue in response to apparently trivial injury, and in this way the anaphylactic reaction is completed."

"Second, death may follow the rupture of a hypoplastic cerebral vessel following an apparently trivial injury. This is possible since Paltauf describes the hypoplastic vessels which show intimal degenerative changes, producing small hemor-

rhages in any situation and occasionally leading to cerebral and other fatal hemorrhages."

DIAGNOSIS.

The diagnosis of thymus hyperplasia is often easily detected by physical examination. Percussion over the sternum reveals a dullness overlapping on each side the ribs and cartilages. It may be felt in the episternal notch. However, I believe the most certain way is by radiography.

Dr. I. E. Liss,⁹ in 1922, conducted a radiographic investigation to determine whether an upper mediastinal shadow usually interpreted as a thymus gland, was obtained in the absence of a thymus hypertrophy, and second, whether the X-ray always demonstrates a hypertrophy of the thymus. Three types of shadows were described, columnar, bulbous and pedicle, with three classes: first, absence of any thymic shadow; second, small thymic shadow, being less than 3 cm. beyond the vertebral transverse processes; third, large shadow, more than 3 cm. beyond the vertebral processes. He found 35 cases in class one; 34 in class two; 50 in class three in infants radiographed 48 hours after birth. After one month he was able to examine 73 of the original 119 and found of class one, 33; class two, 24; class three, 16. He continued the work and found retrogression in size to be greatest during the first two months and almost completed at the end of the second year.

Dr. F. O. Coe¹⁰ found the measurement of the normal supracardiac shadow to be 32 per cent of the transverse thoracic shadow at three months of age and averages 30 per cent at thirty months and stays there up to six years, when it gradually declines to 25 per cent at eight years and remains at about this ratio.

In contrast, the enlarged thymus was found to be 43 per cent at two months, stays at about 45 per cent until the third year, when it descends to just above 40 per cent, and gradually declines to 32 per cent at seven years. He then calls a shadow with a one to three ratio normal and one approaching one to two hypertrophy of the thymus.

Greenthal,¹¹ in an analysis of 2,000 consecutive cases at the University of Michigan Hospital ranging in age from three days to twelve years, reported 90 cases of thymic enlargement, or 4.5 per cent, 87 of which gave no symptoms referable to the thymus.

Many deaths from so-called status lymphaticus have been reported. Dr. L. Hubert¹² reported four cases in 1923; Dr. Wm. C. Carter¹³ reported one following tonsillectomy in 1918; in 1913, Dr. Walter I. Bierring¹⁴ reported four cases in one family. Drs. John Anderson and J. A. Cameron,¹⁵ in the Glasgow Medical Journal of September, 1927, reviewed 100 cases of status lymphaticus and all presented the characteristics described by Paltauf.

Griffith¹⁶ reported seven cases of sudden death in one family strangely suggestive of status lymphaticus. Perkins¹⁷ cited a sudden death under primary anesthesia for tonsil and adenoid operation that had been previously X-rayed and examined clinically, showing no signs of thymus enlargement. At autopsy a thymus weighing 39 gms. was discovered, which lay over the heart and whose shadow was obscured by the heart shadow.

My own independent investigation of thymus hypertrophy was prompted by the very unfortunate accident of a so-called thymic death in my own practice.

The patient, little E. F., age six years, expired as in extreme shock after a simple T. and A. operation, while being lifted from the carrier to the bed. At autopsy it was found to possess a thymus gland weighing 28 gms. On the very morning of the accident two other children of the same family were to have been operated upon. However, operation was deferred until X-ray investigation of the thymus. One child was negative, this having previously undergone a mastoid operation; the third, a second girl, age seven, showed a very large thymus shadow. Needless to say, operation was postponed until after reduction of the thymus shadow by treatment.

In my private series all children under six years of age have been examined by X-ray prior to tonsil and adenoid operation since this mishap. To date there have been 201 children examined with so-called thymus hypertrophy in 26 cases, or 7.7

per cent. Of these, 18 were classified as moderate hypertrophy and 8 as excessive hypertrophy.

It is gratifying to find that my findings so closely coincided with those reported by Dr. Mosher, Macmillan and Motley,¹⁸ who reported finding positive shadows in 7.5 per cent of 4,820 consecutive children of the tonsil and adenoid age.

The following are typical cases of hypertrophy of the thymus which were radiographed and treated with X-ray by Dr. Wm. G. Herrman and later operated on for tonsils and adenoids:

Case 1.—Leonard B., age 2½ years, radiographed May 9, 1927. Treated by X-ray May 12th, again radiographed June 3, 1927; found normal in size and operated upon June 5, 1927. Radiographs appended.

Case 2.—Robert B., age 4 years, radiographed August 12, 1927. Treated August 15th and 26th. Second radiograph on May 22, 1928, still showed a large thymus; treated again on May 25th. The third plate on June 19, 1928, showed a normal thymus. Operated upon June 26, 1928.

Case 3.—Willard P., age 1½ years, radiographed October 3, 1927. Treated April 6, 1928, and May 20, 1928. Second radiograph June 4, 1928, and operated upon June 6, 1928.

Radiographs will not show all cases of enlarged thymus, as Perkins has demonstrated, but with our present knowledge it is our best security against accidental death.

From my studies of the subject matter, I am prepared to agree with Dr. James A. Babbitt,²⁰ who says: "For the purpose of discussion, the premise might be asserted that: status lymphaticus, whether pathologically or physiologically rated, presents a definite menace to operative procedure, and therefore its suspicions should be recognized; its diagnosis should be established; operative decision should be measured with the same or more care than cardiac cases."

CONCLUSIONS.

1. The consensus of opinion is to the effect that between 5 and 7 per cent of all children of the adenoid and tonsil age show a shadow by X-ray suggestive of thymus hypertrophy.

2. X-ray treatment *will reduce an enlarged* thymus with but one or two treatments. Probably the pediatrician has previously warned the parents of the potential danger, thus throwing all responsibility on the shoulders of the laryngologist.

3. Since thymus hypertrophy is a probable menace to the safety of laryngeal surgery in children, and since its presence can, in the vast majority of cases, be demonstrated by radiographs, it would seem that routine examination is a procedure which should be more widely practiced than at present.

BIBLIOGRAPHY.

1. Crotti, Andre: Quoted from Thyroid and Thymus. Philadelphia, Lea & Febiger, 2nd Ed., 1922.
2. Hammar, Johan Augustus: Die Menschenthym in Gesundheit und Krankheit. Leipzig, Akad. Verlagesell., M. N. H., 1926, V. 8.
3. Boyd, Edith: Growth of the Thymus: Its Relation to Status Thymicolymphaticus and Thymic Symptoms. Am. J. Dis. Child., June, 1927, XXXIII, 867.
4. Paltauf: Wiener klinische Wochenschrift, 2nd Ed., 1889, p. 877.
5. Williams, Leonard: The Thymus Gland. N. Y. M. J., CXV, 338, April 5, 1922.
6. Park, Edw. A., and McClure, Roy D: The Results of the Thymus Extirpation in the Dog. Am. J. Dis. Child., XVIII, 317, November, 1919.
7. Plater, F.: Quoted by Ruhräh, John: Pediatrics of the Past. Ed. I, N. Y., Paul B. Hoeber, 1925, p. 239.
8. Symmers, Douglass: The Cause of Sudden Death in Status Lymphaticus. Am. J. Dis. Child., XIV, 463, December, 1917.
9. Liss, I. Edw.: The Thymic Shadow in Infants. Am. J. Dis. Child., XXIV, 192, September, 1922.
10. Coe, F. O.: A Method of Estimating the Size of the Thymus Gland. Am. J. Roentgenol., XV, 222, 1926.
11. Greenthal, Roy M.: The Incidence of Thymic Enlargement without Symptoms in Infants and Children. Am. J. Dis. Child., XXIV, 433, November, 1922.
12. Hubert, Louis: The Enlarged Thymus from the Viewpoint of the Laryngologist. N. Y. M. J., CXVIII, 410, April 4, 1923.
13. Carter, Wm. Wesley: Status Lymphaticus Death Following Tonsillectomy—Autopsy. M. Rec., XCIII, 19, Jan. 5, 1918.
14. Bierring, Walter L.: Status Thymolymphaticus with Report of Four Cases in One Family. Am. J. Dis. Child., VI, 75, August, 1913.
15. Anderson, John, and Cameron, J. A.: Status and Sudden Death. Glasgow M. J., XI, 129, Sept., 1927.

16. Griffith, J. P.: The So-called Thymus Death with an Account of Seven Cases of Sudden Death in One Family. N. Y. M. J., XC, 444, Sept. 4, 1909.

17. Perkins, Chas. W.: Roentgen Studies and Treatment of the Enlarged Thymus in Children. Reprint. N. Y. M. J., CXVII, No. 8, April 15, 1923.

18. Mosher, MacMillan and Motley: A Clinical and Preoperative Study of the Thymus in Children of the Tonsil and Adenoid Age. The Laryngoscope, XXXVI, 1, January, 1926.

19. Perkins, Chas. W.: X-ray Study of Five Hundred Children for Thymic Enlargement. Am. J. Roentgenol, XV, 216.

20. Babbitt, James A.: The Diagnostic Problem in Status Lymphaticus. Arch. Otolaryng., Vol. I, No. 2, February, 1923.

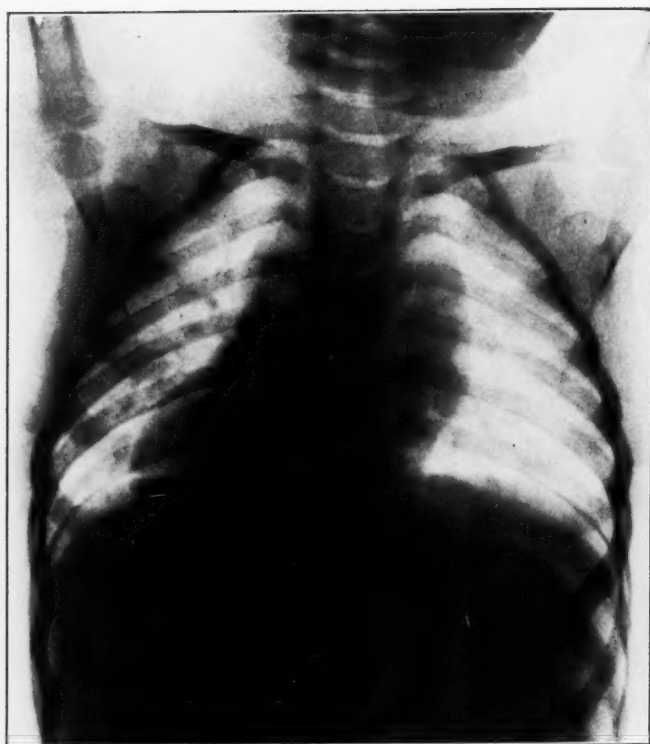


Fig. 1. Case 1. L. B. Before treatment.



Fig. 2. Case 2. L. B. After treatment.

XXIX.

THE DIAGNOSTIC PROBLEM IN ORBITAL
CELLULITIS.*

By JAMES A. BABBITT, M. D.,

PHILADELPHIA.

It would be more conventional to present before this body some original research or refinement in operative technic than a theme of such routine experience and one perhaps already too much weighted with fixed opinion. Even the title presents some challenge. The orbital cellulitis due to direct extension from ethmoid, frontal or other sinuses, the simple edema from block in ophthalmic venous drainage and the somewhat complex symptoms of matured orbital abscess, have so much in common that they are frequently interpreted in terms of the other.

The purpose of this paper is to invite diagnostic discussion of this somewhat baffling condition, which has little prospect of spontaneous recovery and the inevitable one of extension if not impeded, requires a nicety of operative decision, and when combined with a true exophthalmos offers in perspective the ominous threat of cavernous sinus thrombosis. It will review the pathologic background of orbital cellulitis and cavernous sinus thrombosis, contrast the symptoms in these two types of orbital invasion, present a brief description of four selected cases and venture some conclusions.

Orbital cellulitis, as its name implies, is an inflammatory involvement of the soft tissues of the orbit. Such could occur from direct traumatic injury and one of the cases later cited will indicate such complication. The greater majority suggest infective origin and usually from the adjacent accessory nasal sinuses. Traumatic injuries and extension from general facial infection, such as erysipelas, present less confusion in diagnosis and may be omitted from this consideration.

*Read before the American Laryngological, Rhinological and Otolological Society, Inc., May 30, 1930.

Since the close of the last century the conception of orbital cellulitis has steadily changed. No longer do the idiopathic and metastatic views pertain, and the term septic thrombophlebitis has become a common one. Coincident with this change has developed a new interpretation of focal infection in the deeper sinus areas, perhaps nonpathologic to routine nasal examination. Faulkner,¹ in a recent contribution, states "the condition formerly described as orbital cellulitis is usually direct extension from frontal or ethmoid sinus," and in a later note, "extension of infection causing marked swelling of orbital tissue with exophthalmos may occur from sinusitis though no pus can be demonstrated in the orbit at the time of operation." The hyperplastic turbinate membrane and the slight clouding in ethmoid cells with thickened walls, diagnosed by skillful roentgenology, have obtained a new importance. The nasopharyngoscope may confirm the delicate stream of exudate in middle fossa or about sphenoid ostia, and surprising relief result in various infective processes about the eye, from complete exenteration of sinus tissue which appears but mildly diseased. The intimate relation of optic nerve to sinus walls and dangerous contiguity of other eye structures have been discussed at length by White,² Ballenger,³ Crane⁴ and others. They have demonstrated the frequency of toxemia as well as actual sepsis—the rather constant immunity is more surprising than the actual occurrence of infection.

According to Hajek,⁵ there are four possibilities in the extension of diseased process from the bony framework of the sinuses:

1. Extension of inflammatory process through apparently intact bone.
2. Extension by thrombosis of one of the large veins perforating bone and spreading infectious material to the other side.
3. Direct extension of the ulcerating process to the bone with periostitis, osteitis and finally necrosis with perforation.
4. Extension of inflammatory process through dehiscence in the bony wall.

Whether this be accepted in whole or part, it should warrant a more frequent surgical attempt to reach the source.

When there is definite protrusion of the eyeball in connection with orbital cellulitis, the decision between orbital abscess and cavernous sinus involvement complicates the simple problem of extension. Vail⁶ has reported four cases of exophthalmos from sphenoid, posterior ethmoid, anterior ethmoid and frontal sinus involvement, respectively, where an opening was found in the periosteum of the orbital wall, eliminating, in these at least, the theory of access to orbital veins of an infectious current draining contiguous accessory sinus.

In considering the anatomic and pathologic background of cavernous sinus thrombosis, the more serious diagnostic problem linked with orbital cellulitis, emphasis should be put upon the rarity of authentic reported cases. Dwight and Germain,⁷ in 1902, gave the number reported as 182, and Smith,⁸ in 1918, less than 300. Chisholm and Watkins⁹ found only eight cases recorded in the Johns Hopkins Hospital records of 50,000 surgical cases from 1889 to 1919. This almost rivals agranulocytosis in paucity of record.

The cavernous sinuses rest beside the body of the sphenoid bone, connected by the circular sinus which surrounds the pituitary body. They lie in intimate relation to the optic nerves and chiasm, the sphenoid sinus and gasserian ganglion. The superior and inferior ophthalmic veins empty into the cavernous sinus of each side, respectively, and the sinus, in turn, terminates in the superior and inferior petrosal. On the lateral walls lie the third, fourth, ophthalmic and maxillary divisions of the fifth and just within, the sixth nerve. The internal carotid passes through it. Veins from the dura, sphenoid sinus and pituitary are received by it, and emissary veins go out through basal foramina to connect with pterygoid and other venous networks. St. Clair Thomson¹⁰ and Eagleton¹¹ have most adequately portrayed the nature of the resultant sinus infection, a septic thrombophlebitis, starting with a septic infiltration of the venous walls, then endophlebitis with coagulation of blood along the line of inflammation until the lumen is obliterated, fibrinous change and adhesion to walls, clot breaking down into a semipurulent mass and portions thrown off into the circulation, with thrombi in other veins.

With the rich field of circulatory connection in this anatomic review, infection advent is readily explained. Cases aris-

ing from the upper lip and alveolar margin, auriculomastoid area via the petrosal sinuses, or the great distribution of the facial veins, concern the title of this paper less than those having origin in the accessory sinuses. Sphenoid sinus extension is perhaps rare, but it is interesting to note that in the Chisholm-Watkins⁹ series of twelve reported cases, four were assigned to focus in paranasal sinuses, two to facial infection and one to orbital abscess. In Loeb's¹² group of nine cavernous sinus thrombosis fatalities recorded in his later survey, five followed intranasal operative work. Perhaps to clarify the possibility of extension from initial orbital cellulitis, more emphasis should be placed upon the many anastomotic possibilities of the facial vein and cavernous sinus through the angular, ophthalmic, nasal, supra- and infra-orbital and pterygoid plexus of veins.

Expressed most simply, the problem in orbital cellulitis is somewhat as follows: A case presents with marked, rather uniform swelling in the orbital cavity—this may appear without much premonition and is of acute inflammatory character, with more or less extension of edematous swelling to surrounding parts, cheek, temple, nasal border and frontal region. In a considerable percentage of cases the eye seems pushed forward in its socket, a true exophthalmos. Bulbar and palpebral conjunctivæ are swollen and edematous, and there is often marked ptosis of the upper lid. The eye may be rotated upward, outward or downward from inflammatory pressure of the adjacent sinus involved. The eye is acutely sensitive to light and pressure, and the patient, if not too ill, complains bitterly of pain. Sight may be diminished, and this is most probably due to the swelling, for fundus examination reveals little more than swelling and increased tortuosity of retinal veins. In orbital cellulitis there should be little tendency to papilledema or functional disturbance of third and sixth nerves. Orbital swelling has the tawny, semi-indurated feel of cellulitis, may have some discoloration and often tends to creep over to the nonaffected side. There may be actual edema around the other eye. Septic evidence may be marked, with high fluctuating temperature, chills, sweats and delirium.

The syndrome of cavernous sinus thrombosis, according to Chisholm and Watkins,⁹ comprises three groups of symptoms:

(1) Those due to venous obstruction; (2) those due to involvement of neighboring nerves, and (3) those due to general sepsis. In the first group belong the postorbital distension producing exophthalmos, edema of lid and nasal bridge, intra-orbital congestion noted in retinal edema, dilatation of veins, clouded media and sometimes change in disc; to the second, ptosis when due to paralysis of oculomotor, dilatation of pupil, fixation of the eyeball, pain, supraorbital headache and occasional loss of vision. The septic symptoms of the third group are conventional, with early chills and frequent meningeal indications.

Compare these with conditions in severe orbital cellulitis. Exophthalmos and extension to other eye occurs in both. It is difficult to separate the ptosis of paralysis from that of inflammatory edema, rigid position of the eyeball due to paralysis of the third, fourth and sixth nerves from the fixation due to engorgement of adjacent tissue. Intraocular changes, pain and its reference, failure in vision, symptoms of toxemia or profound sepsis and laboratory blood studies are relatively alike, with possible exception of high leucocyte count and blood stream infection.

Three of the cases presented here for discussion were tentatively diagnosed cavernous sinus thrombosis; one in autopsy proved negative and the other two recovered. The 7 per cent recovery without operation⁹ must always raise the question of diagnosis without autopsy.

Case 1.—A. S., boy of 11, was brought down from summer camp in September, 1923, with typical picture of orbital cellulitis of the right eye, temperature range around 103, moderately septic, leucocyte and differential not alarming, but orbital swelling extreme. The left eye was closed; edema spread into face and over toward the opposite eye. Pain was intense to both pressure and light. The striking feature of this case was that repeated intranasal and fundus examinations, confirmed by consultations of authority, showed nothing abnormal. The frontal and ethmoid findings appeared to be only due to the extreme cellulitis and hemorrhagic discoloration.

but on the sixth day symptoms became so profound as to demand operation. Entrance was made through the frontal sinus. A postorbital abscess was found which had eroded through the posterior part of the frontal sinus floor and several drams of pus were evacuated. During convalescence the superior orbital broke down, requiring plastic correction some years later, which was done by Dr. Frazier. This case presented much that pointed toward cavernous sinus thrombosis, but was promptly relieved by drainage of orbital abscess. In the light of subsequent experience, this case clearly indicated intranasal sinus surgery at an early stage, despite the absence of local symptoms.

Case 2.—H. R., Jr., a young man of 20. Writer saw in consultation in a neighboring city in March, 1928, this case of marked orbital cellulitis of the left eye. A few days before onset the patient had a boil on the left side of the chin, followed by a sty on the left eye. Onset was most severe, with septic temperature, frontal headache, exophthalmos, swelling extending to the opposite side. As X-ray studies showed involvement of left maxillary antrum, ethmoid and frontal, the otolaryngologist in charge, four days after onset, performed ethmoidectomy, enlarged the nasofrontal duct, punctured antrum beneath the inferior turbinate and, to obtain more room, did partial submucous resection of septum. The patient improved for 36 hours. Blood culture eighth day after onset showed staphylococcus albus. On the ninth day temporofrontal intracranial complications appeared and spinal fluid showed a few cells with gram positive encapsulated intracellular diplococci. Nine days after onset the patient was examined by a neuro-surgeon, Dr. Grant, whose findings included right hemiplegia with positive Babinski, and an almost complete aphasia which had developed in past eight to twelve hours. Tobey-Queckenstedt test suggested positive reference to left lateral sinus. Leucocytes had risen from 13,000 to 26,000. Spinal cell count was 125 per cu. mm. Impressions at this time were that the boy had a left frontal lobe abscess and probably cavernous sinus thrombosis. Operative interference was refused. Patient died on the eleventh day. The writer attended the autopsy, which revealed a thick sub-arachnoid collection of pus over the left frontal region and

frontotemporal region. There was absolutely no sign of cavernous or left lateral sinus thrombosis.

It is needless to comment on the diagnosis problem in this case.

Case 3.—Dolly H., age 4 years. Admitted to Children's Hospital ward, October 25, 1929. The child presented these symptoms: Unconsciousness, orbital swelling of the left eye, profuse nasal discharge and high fever. She had been treated in dispensary for sore throat and upper respiratory infection two days before. Early this morning she called for water and an hour later could not be aroused.

Clinical examination showed marked cellulitis of left orbit and ptosis of upper lid with marked edema. Intranasal examination by nasopharyngoscope showed pus coming down in quantity in middle meati of both sides, more on the left. X-ray studies showed involvement of ethmoids and antra, both sides, also more on the left. Blood examination reported leucocyte count of 20,000 and neutrophile 70. Blood culture had no growth in seven days. Nasal pus culture showed staphylococcus aureus the predominating organism; ophthalmologic examination was practically negative. This child was profoundly sick, with extreme pain about left eye and continuously comatose. Consensus of opinion was that this case was probably in the initial stages of cavernous sinus thrombosis but that intranasal sinus drainage was indicated. On the day following admission, under general anesthesia, both ethmoid areas were exenterated and a large window opening was made in the left antral wall beneath the inferior turbinate. This child made a reasonably prompt recovery after operation.

Case 4.—Benj. H., age 6 years. Admitted to Children's Hospital February 6, 1930. Clinical picture on entrance was that of a marked orbital cellulitis of the right side. Edema extended over the bridge of the nose and to surrounding parts of the face. The right eye was completely closed with marked edema of both lids and little motion. The impression was that of orbital cellulitis or cavernous sinus thrombosis.

Patient reported being struck in the right eye in play the day before entrance. In the evening had intense pain over right temporoparietal region. He reported mild headaches three times in the past month but no disturbance of vision, dizziness or photophobia. Two days after admission edema and redness seemed somewhat diminished but chemosis and exophthalmos increased. Pupil was dilated and the patient was unable to see, probably on account of swelling. In the afternoon some puffing and redness of the other eye was noted.

In the afternoon of the second day after admission, the patient had a chill with temperature of 107 and two subsequent chills. Blood culture was taken and showed staphylococcus aureus. X-ray examination indicated clouding in the right ethmoid and antrum. On account of the writer's convalescence from recent illness, operation was performed by Dr. Campbell, associate in the department. The right ethmoid was entered by the external route and pus was found in the inner side of the orbit, with necrosis of inner orbital plate adjacent to ethmoid cells. Diseased anterior and posterior ethmoid cells were removed but no definite postorbital abscess was found.

The child had a stormy recovery. Blood transfusions and vaccines were given. Ten days after admission metastasis of infection occurred in the right humerus and a month later in the right hip. Both of these areas, osteomyelitic in appearance, required operation. The inferior orbital swelling required opening and drainage. Child is doing well but not yet out of the hospital.

This condition showed such marked resemblance to cavernous sinus thrombosis that the various methods of radical surgery were duly considered. Slides will demonstrate the appearance of this and the previous case.

The problem of this paper is primarily the diagnosis in borderline cases between orbital cellulitis and cavernous sinus thrombosis. It is the field of both otolaryngologist and ophthalmologist and their combined efforts may fail. This diagnosis requires analysis of factors in etiology, interpretation of coincident localized or general meningitis, and may determine the question of surgical intervention.

CONCLUSIONS.

1. The so-called orbital cellulitis is a secondary manifestation of infection and is a symptom complex rather than an entity.
2. Recent opinion emphasizes its accessory sinus origin, save in cases of obvious external cause.
3. Such extension occurs through venous and lymphatic channels, dehiscence in wall or osseous necrotic change.
4. Symptoms of cavernous sinus thrombosis and orbital cellulitis are often so identical, even in degree, that diagnosis is difficult.
5. A concluded diagnosis will frequently be reversed by autopsy findings.
6. The low percentage of recovery under radical procedure and the serious prognosis without interference justifies initial nasal sinus surgery, even in cases of doubt.

1912 SPRUCE STREET.

BIBLIOGRAPHY.

1. Faulkner, E. Ross: "Inflammatory Affections of Sinuses," page 92, of Jackson-Coates' "The Nose, Throat and Ear and Their Diseases," 1929.
2. White, Leon E.: "The Etiology and Pathology of Loss of Vision from the Accessory Sinuses." Trans. A. L. R. and O., 115-131, 1921.
3. Ballenger, W. L.: "Diseases of the Nose, Throat and Ear," third edition, page 816.
4. Crane, Claude G.: "The Anatomical, Pathological and Clinical Relationship of the Posterior Sinuses to Optic Neuritis." Trans. A. L. R. and O., 176-210, 1926.
5. Hajek, M.: "Nasal Accessory Sinuses," vol. I, page 107.
"Nasal Accessory Sinuses," vol. II, page 437.
6. Vail, D. T.: "Types of Orbital Abscess and Exophthalmos Due to Intranasal Suppurative Processes." Trans. A. L. R. and O., 244-251, 1919.
7. Dwight, E. W., and Germain, H. H.: "Thrombosis of the Cavernous Sinus, with report of four cases, including one cranial operation." Boston M. and S. J., 146:456, 1902.
8. Smith, D.: "Cavernous Sinus Thrombosis, with Notes of Five Cases." Arch. Ophth., 47:482 (Sept.), 1918.
9. Chisholm, J. Julian, and Watkins, S. Shelton: "Twelve Cases of Thrombosis of the Cavernous Sinus." Archives of Surgery, 483-512, Nov., 1920.

10. Thomson, St. Clair: "Causes and Symptoms of Thrombosis of the Cavernous Sinus." *Ophth. Rev.*, 27:296, 1908.

11. Eagleton, Wells: Proceedings of the Section on Otology and Laryngology. Trans. Coll. Phys., Philadelphia, 1924.

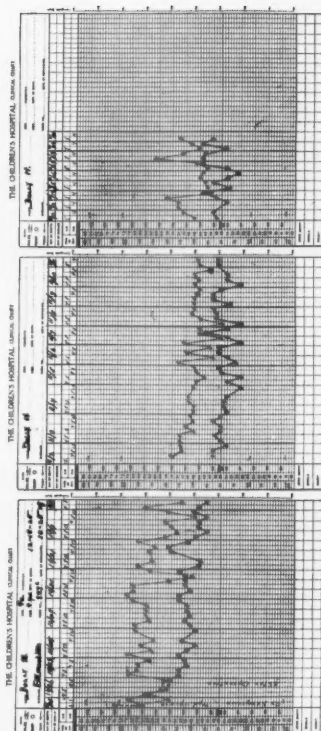
12. Loeb, Hanau W.: "Fatalities Following Operations upon the Nose and Throat not dependent upon anesthesia: A Study of Three Hundred and Thirty-two hitherto unreported Cases." Trans. A. L. R. and O., 168-190, 1922.



Case 3.—Preoperative.



Case 3. Postoperative.



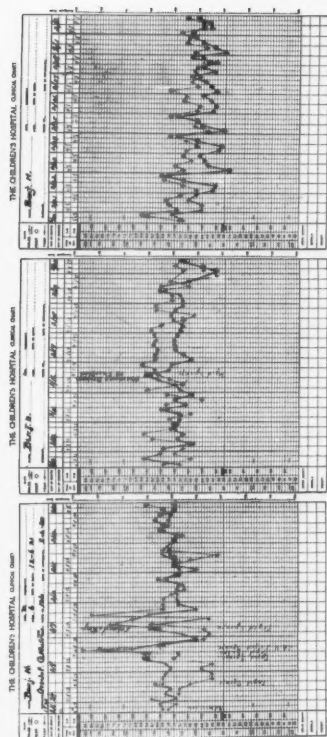
Case 3. Temperature Chart.



Case 4. Preoperative.



Case 4. Postoperative.



Case 4. Portion of Temperature Chart.

XXX.

PALATAL PALPEBRAL REFLEX.*

BY CHARLES J. IMPERATORI, M. D.,

NEW YORK.

In presenting this clinical observation, which seems to be an original one, before this Society for its consideration, it is done so entirely in the spirit that this contribution needs further investigation and study. Original observations sometimes are not as new as the observer thinks, and should this contribution fall within these limitations your forgiveness is asked.

DESCRIPTION OF THIS REACTION.

This reflex is one that is observed as a normal reaction. It consists of a blinking of the upper eyelids on stimulation of the soft palate. Its absence, excepting in rare instances, may be considered abnormal. There are some few persons who normally seem to possess no reaction. They are usually the type who will permit extensive examination of the nasopharynx without interference.

METHOD OF INDUCTION.

The reaction is induced by a percussion of the soft palate. This is done with a small stick applicator, and the point of the percussion stick should be applied to the soft palate midway between the midportion of the uvula and the anterior pillar where it merges into the soft palate.

The reaction is bilateral and only occasionally on the side of percussion. Frequently after one or two attempts at induction of the reflex there will be a reaction in which the patient jerks the head backwards. At times, on induction of the reflex, an expression of fear will come over the patient's face. At no time has there been any vomiting reflex induced in attempts at this reaction. Stimulation of the inner side of the cheek,

*Presented before the Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, May, 1930.

tongue, hard palate or base of the tongue does not elicit this reflex.

The type of palate that gives the reaction best is one that is broad and that is some distance from the posterior pharyngeal wall. This type of palate on percussion in the place indicated will give a marked blink on the side struck and a slight reaction to a complete blink on the opposite side. Those palates that have been deformed by tonsillectomies, if percussed near the base of the uvula, give the reaction just as well as those in whom no operation has been done. Small palates that are very closely applied to the posterior pharyngeal wall, as a rule do not give as marked a reaction as those described above.

DISCUSSION OF POSSIBLE MECHANISM.

¹"According to Herrick, a reflex act, as this is termed, is usually defined by physiologists as an invariable, mechanically determined adaptive response to the stimulation of a sense organ, involving the use of a center of correlation, and the conductors necessary to connect this center with the appropriate receptor and effector apparatus. The act is not voluntarily performed, though we may become aware of the reaction during or after its performance."

²It is an inherent function of the nervous organism and is already present at birth, and must be distinguished from acquired automatisms. It has been suggested that instincts are, in reality, highly complex chain reflexes.

Reflexes serve as valuable indicators of the health or disease of various parts of the nervous system and, as a result, great importance attaches to the presence or absence of the pupillary reflexes, the knee jerks, the plantar reflexes and many others. The reflex arcs may be located in widely different parts of the central nervous system. The simpler ones are in the spinal cord, some of the more complicated ones, such as those serving the reflex movement of respiration, are localized in the hindbrain. Others again, such as those governing the reflex movements of the heart and blood vessels, involve the sympathetic ganglia. In the newborn infant all movements are either reflex or automatic, the cerebral cortex comes into play as the infant gains and adapts itself to its environment, necessitating

the inhibition of certain reflex acts and the initiation of voluntary movements.

There are three types of reflexes, the deep or tendon reflexes, the superficial or cutaneous, and the visceral.

³The arc may be through an afferent arm, the trigeminal nerve, which bears the impulses to the substantia gelatinosa and by collaterals to the facial nucleus whose efferent arm the facial nerve brings the impulses to the eye muscles.

There is also the possibility that part of the arc is through the ninth and remotely through the eleventh nerve. Byron Stookey, in an article on glossopharyngeal neuralgia, seems to prove that the soft palate is supplied by the glossopharyngeal nerve. This patient was one in whom there was an intractable neuralgia of the soft palate, and by section of the ninth nerve he induced a total paralysis of half of the palate. There did not seem to be any sensory paralysis in this patient following the operation.

In any event the afferent arm of the arc would seem to be rather a complicated mechanism, and the exactness of these anatomic details are merely suggested. Further study must be given to this before any definite assertions can be made. In an analysis of the movement of the eyelid, the various moving picture illustrations show that the closure of the eyelid is very rapid in comparison to the opening, approximately four to one. In other words, the interval between the application of the stimulus and the closure of the eye is one-quarter less time than the gradual opening of them. (See illustration.)

DESCRIPTION OF PATHOLOGIC CASES CITED.

In those patients with a facial paralysis percussion of the soft palate on the side of the paralysis does not produce an eye blink. Occasionally there will be a blink of the eye on the sound side. This always occurs if the palate is percussed near the base of the uvula. If the sound side is percussed we get a blink in the nonparalyzed eyelid. In a patient with a sensory disturbance in the palate and one in whom there is a total loss of sensation on the same side of the body, on percussion, there was a definite and positive blinking of both eyes and more marked on the side percussed. This patient did not regurgitate

fluids through the nose on swallowing. Unfortunately, no cases of paralysis of the soft palate following diphtheria have been observed.

OBJECT OF THE REFLEX.

1. To determine the tonus of the musculature of the soft palate.
2. Slight paralyses of the soft palate are easily determined by this reflex.
3. Confirmatory between a sensory and muscular paralysis of the soft palate.
4. Definitely absent in paralysis of the soft palate.

CONCLUSION.

It is possible that there are two elements in this reflex, that of a deep or muscular one, and secondarily, a sensory one. It would seem, from the cases examined, that the muscular element was the most important one. This reflex may be classified as a deep one. The study of its absence I feel will be of distinct value to all, and particularly so to laryngologists and neurologists.

BIBLIOGRAPHY.

1. Sachs, B., and Hausman, L.: *Nervous and Mental Diseases*. N. Y., 1927, Herrick, p. 11.
2. Sachs, B., and Hausman, L.: *Nervous and Mental Diseases*. N. Y., 1927, p. 105.
3. Tilney and Riley: *The Form and Function of the Central Nervous System*. Hoeber, N. Y., 1923, p. 387.



The photographs A and B are continuous photographs and are not spliced. It was necessary to cut and splice the film in Column C, because this was an illustration taken from Dr. Deaver's Anatomy. The speed of the camera that took these photographs was less than a 25th of a second per exposure. Illustrations 4 in Columns A and B show the eyelids open and the stimulus about to be made to induce this reflex. Illustrations 5 in Columns A and B show the eyelids gradually opening. In other words, the interval between applying the stimulus and the closure of the eyes is one-fourth less time than the gradual opening of them. Column C shows where the stimulus is applied.

XXXI.

PSAMMOMA (BRAIN-SAND TUMOR) REMOVED
FROM THE ETHMOID AND SPHENOID AREA.*

BY HENRY M. GOODYEAR, M. D.,†

CINCINNATI.

Psammoma, or "brain-sand" tumors, are of sufficient rarity to justify their being placed on record. Their recognition and removal from the ethmoid area may be accomplished with considerable difficulty and hazard.

In 1867, Virchow coined the term "psammoma" (sand-tumor), peculiar to the meninges of the brain and spinal cord. Piersol said they were practically normal in the adult pineal gland in the form of a so-called "brain-sand." They are found particularly, however, in connection with chronic inflammation and tumor formation, sometimes sarcomatous or endotheliomatous in character.

Korsner, in his "Human Pathology," describes a psammoma as a tumor made up of many small psammatous bodies of concentrically laminated calcareous deposit, usually microscopic in size, but sometimes obtaining a diameter of two to three millimeters.

The case which I am about to describe had a psammoma which apparently originated in the region of the cribriform plate and extended up into the left region of the cerebrum and down into the nose.

When the patient was admitted upon the nose and throat service at the Cincinnati General Hospital he was complaining of severe headache and rapid loss of vision. These symptoms were of only ten days' duration. The vision was 20/30 and the field contracted.

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Eight months previous to this time the patient had been on the general surgical service, complaining of severe left frontal headache and gradual loss of vision for one month.

At this time Dr. George Heuer removed a tumor from the left frontal area of the cerebrum. As the tumor had extended through the dura in the region of the cribriform a leak of spinal fluid was inevitable after operation. This was avoided, however, by carefully suturing a piece of fascia lata in the dura opening where it lived, and no evidence of cerebrospinal rhinorrhea ever occurred.

The outer shell of the tumor was bony hard, but the inner portion could be easily carved. It was considered to be epitheliomatous in character. The vision returned eight hours after operation and there was no particular discomfort or disturbance of vision up to his present illness.

Intranasal examination revealed a hard tumor mass apparently growing out from the septum and completely filling both sides of the nose down to a level of the inferior turbinates.

Roentgenograms showed a dense clouding of the ethmoid and sphenoid areas on both sides and a deep clouding of the left antrum.

Taking advantage of the absence of the left eye, which had been removed sixteen years before, following a traumatic injury, a wide opening was made into the nose by a Killian incision and the removal of a large portion of the frontal process of the maxilla.

Through this opening a bony tumor which could be easily carved was pared away. The mass extended so far laterally that the ethmoid labyrinth with the lamina papyracea were removed on both sides as well as what remained of the cribriform plate. There was no escape of spinal fluid. The sphenoid cavities were large and completely filled with the bony growth without any definite line of demarcation. The medial wall of the left antrum had disappeared, and the sinus cavity was found to be filled by an extension of the tumor mass. The greater part of the nasal septum was removed.

Iodoform packing was used to control rather severe bleeding in the sphenoid area. The vision rapidly returned within twenty-four hours, and the headaches have not returned, nor

has there been any evidence of return of the tumor tissue within the nose one year after operation.

A psammoma probably always originates from the dura, and when it extends through this structure and through the cribriform plate it is evident it would be most difficult to remove without a resulting dural fistula and loss of spinal fluid. The preceding intracranial operation in this case and the fortunate closure of the dura by an insertion of fascia lata made the nasal operation possible.

A psammoma in the ethmoid area might be most commonly confused with an osteoma, since osteomata seem to have some predilection for this region and often extend into the ethmoid area, the orbit and the frontal sinus. I reported one such case in 1925.*

Eight weeks ago I removed a tumor, suspected to be a psammoma, from the ethmoid area in a young woman who was complaining of severe headaches and disturbance of vision.

The tumor apparently grew from the upper portion of the septum and extended through the ethmoid region and into the right orbit. By a Killian incision the mass was completely removed, with a wide exposure of dura. Above the bony tumor mass was a large collection of pus, and the floor and posterior wall of the right frontal sinus had been completely absorbed. On microscopic section the growth proved to be an osteoma rather than a psammoma.

Several years ago I operated upon an endothelioma in this area. Through an external opening the dura was elevated and the anterior two-thirds of the cribriform plate removed. There was no escape of spinal fluid, and the softness of the tumor distinguished it grossly from a psammoma or osteoma.

556 DOCTORS' BLDG.

REFERENCES.

Bland-Sutton, J.: The Choroid Plexus and Psammomas. *Brit. M. J.*, 1922, Vol. 1, pp. 213-14.

Delafield and Prudden: *Text-book of Pathology*. P. 859, Ed. 8, N. Y., 1907, Wood.

*Goodyear, Henry M.: Osteoma of the Frontal Sinus Extending into the Orbit and Anterior Cerebral Fossa. *The Laryngoscope*, October, 1925.

Durck: Über Psammoma Endotheliome der Dura Mater. Münchener med. Wochenschrift, 1907, V. 54, p. 1154.

Von Eicken: Psammoma of Ethmoid Sinus. Schweiz. med. Wochen., 1922, Vol. 52, p. 495. Abstract J. A. M. A., 1922, Vol. 79, p. 413.

Ernst, Paul: Über Psammome. Beiträge z. Path. Anat. (Ziegler), 1892, Vol. 11, pp. 234-59.

Ewing, J.: Neoplastic Diseases. P. 479, Ed. 3, Phila., 1928, Saunders. Faure-Beaulieu, and Martel, T. D.: Case of Paraplegia Cured by Removal of a Psammoma. Bull. et Soc. Med. Hop. de Par., Vol. 48, p. 979, 1924.

Goodhart, J. T.: A Large Psammo-Sarcoma (2 in. by $1\frac{1}{2}$), Growing from the Angle Formed by the Tentorium and Falx Cerebri. Tr. Path. Soc., Lon., 1885-6, Vol. 37, p. 16.

Jadasshon: Psammoma of Occiput. Schweiz. med. Wochenschrift, Vol. 57, p. 42, 1927.

Karsner, H. T.: Human Pathology. P. 105, Phila., 1926, Lippincott.

Müller, E.: Fall von Psammome der Dura Mater. München. med. Wochenschrift, 1918, Vol. 65, p. 141.

O'Kelly, W. D.: Notes on a Psammoma of the Dura Mater. Dublin J. Med. Science, 1921-22, pp. 26-27.

Targett, J. H.: Psammoma of the Dura Mater. Tr. Path. Soc., 1895-6, Lond., Vol. 47, p. 3.

Woolcombe, W. L.: A Case of Psammoma of the Pituitary Body, with Remarks as to the Function of That Structure. Brit. Med. J., 1894, Vol. 1, p. 1351.

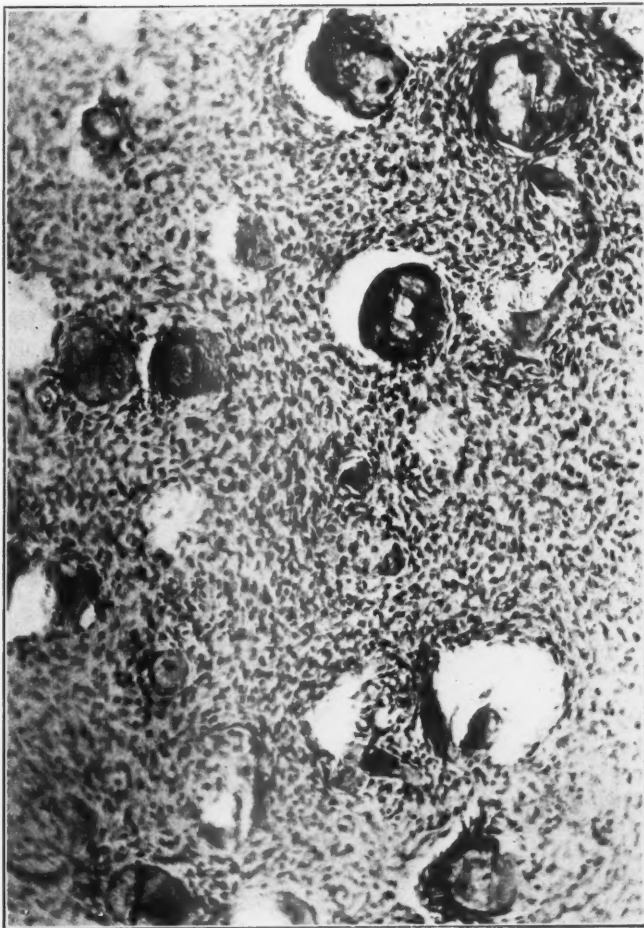


Fig. 1. Microscopic section (low power) from the tumor removed as recorded herewith showing the numerous psammomatous bodies of concentrically laminated calcareous deposit.

XXXII.

SOME NOTES ON PROTEINS AS THEY APPLY TO OPHTHALMOLOGY AND OTORHINO- LARYNGOLOGY.

BY SANFORD R. GIFFORD, M. D.,*

CHICAGO.

The study of the human body revolves around the proteins, since these form the substance of the living body of all plants and animals and exist in every cell. The most important characteristic of proteins in general is the large size of the protein molecule, and it is to this large and complicated molecule that the proteins owe the properties which make them essential to the animal mechanism. Liebig's formula for albumen was $C_{216}H_{328}N_{54}S_2O_{108}$ the molecule being 4,870 times as large as the hydrogen molecule. It is now believed that this was too simple, and Lloyd gives as the molecular weight of gelatin, one of the simplest proteins, 10,000, while that of fibrin is about 42,000, hemoglobin 50,000 and casein 192,000. All proteins contain carbon, hydrogen, nitrogen and oxygen and some contain also sulphur and phosphorus. How these elements, combined by molecular aggregation from their inorganic forms in the primeval slime to produce proteins, is the problem of life itself.

The vegetable proteins are relatively few and simple, since plants use sunlight to synthesize cellulose and other compounds out of carbohydrates, while the animal organisms must use proteins for its supporting structures and for an infinite variety of purposes. Thus we require various proteins. The simple proteins include the albumins, of which serum albumin is an example; the globulins; the albuminoids, which as elastin, collagen and keratin, make up most of our supporting structures and the skin; histones, which are found in the gland cells; and protamines, which occur chiefly in the sperm cells.

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The simple proteins are composed only of amino acids. The conjugated proteins are combinations of proteins with a non-protein element. Thus the mucoproteins, used for lubrication of the food and air passages, include a carbohydrate group; the chromo-proteins various colored bodies, such as hematin; and the nucleo-proteins are combinations of proteins with nucleic acid.

The properties of the various proteins depend on the particular amino-acids present in each and the way these are linked. Gelatin, for instance, lacks tyrosine and tryptophane, and hence is not an adequate source of protein for nutrition.

The presence of an amino (NH_2) and a carboxyl group (COOH) in the amino-acids gives to the body proteins their property of combining with both acids and bases in the tissues so that they may act as buffers, and in conjunction with the inorganic salts present, keep the body fluids to a reaction near enough neutrality to support life. Each protein is characterized by a certain point, called its isoelectric point, at which it acts neither as an acid nor a base, combining with neither acid or basic radicles. For gelatin, for instance, this is a H. ion concentration of P. H 4.7. In solutions more alkaline than this it will combine with silver, and in solutions more acid with bromine. On either side of the isoelectric point a protein absorbs water and swells. In the body cells under normal conditions the reaction is never far from the isoelectric point of the cell proteins.

This faculty of absorbing water and swelling is due to another property of the proteins, that of forming colloidal solutions in water. The protoplasm of cells, the blood serum and intercellular fluids are all such colloidal solutions, and it is to the relatively new science of colloidal chemistry that we must look for an explanation of their behavior. In a colloidal solution the molecules are distributed so that they cannot move freely, as in a true solution, being aggregated in particules from .1 μ to 1 μ , in diameter. This aggregation prevents them from dialyzing through a parchment membrane, in distinction to solutions of crystalloids, which will so dialyze.

The phenomena of tissue metabolism cannot be explained satisfactorily in terms of true solutions of proteins in water, since the osmotic pressure, for example, of protoplasm is from

five to thirty-five times what would be calculated from its chemical content, and its electrical resistance is correspondingly higher. In such a system the addition of certain crystalloids, among them K Cl, decreases the electrical resistance, as these are known to decrease the resistance in tissues and to increase the permeability of the cell membranes. It is probably by such an effect on the cell membranes that we are to understand synergism, the increased effect of certain drugs when salts, themselves inactive, are combined with them. Thus, as Antonibon has recently shown, the speed and efficacy of 1 per cent atropin when used by instillation for mydriasis may be increased 100 per cent by adding 1 per cent K Cl, although this salt itself has no effect on the pupil.

It is through their effect on the colloidal systems of the body that we must look for an explanation of the effect of many therapeutic measures. What happens, for instance, when we introduce a foreign protein into the body to combat an inflammation for which we have no specific therapy? According to Petersen, in his "Protein Therapy and Non-specific Resistance," the harmful effects of bacterial infection are due to the split products of proteins formed in the bacteria themselves or from tissues which the bacteria attack. These split products, histamine and other peptides and amino-acids, are extremely toxic, and successful resistance to infection depends on the ability of the tissue to dissolve the bacteria and break down their toxic products to simpler nontoxic forms. Although in certain diseases specific antibodies are formed which bind the toxins or agglutinate the bacteria, or make them susceptible of phagocytosis, a chief means of defense in any infection is the production of enzymes, protease and ereptase, which break down the foreign protein and its products. The tissues are prevented from lysis by another form of enzyme, anti-ferment, which is present in the serum and increased during infection. Injection of a foreign protein produces an immediate decrease in the anti-ferment and a marked increase in the enzymes protease and ereptase. Necrotic tissue and bacterial cells are broken down, first into split products which are toxic, resulting in the symptoms of increased temperature, malaise, etc. These toxic products then split to simpler nontoxic forms and the temperature drops. This mobilization of

enzymes is due to an effect on the colloid system of the cells, the exact nature of which is unknown. At the same time the cell membranes become more permeable, probably by a change in the phase of their lipoids. Any specific antibodies already formed in the cells and attached to them are thrown into the circulation where they exert their specific effects. The primary leukopenia which occurs after protein injection is due, according to Petersen, to a collection of blood cells in such organs as the spleen, where bacteria are especially numerous. This is followed by a leucocytosis which is undoubtedly of importance in combating infection. The increase of enzymes in the serum was found by Jobling and Petersen to last for three to four days. The leucocytosis, according to Wick, also lasts three to four days, and his best results were obtained when an interval of four free days was allowed between injections. Wick believes the leucocytosis is the important factor in the reaction and found that this was often obtained without fever. Petersen considers that leucocytosis is only one of the phenomena, which with fever, mobilization of enzymes, increased cell permeability and vasodilatation, represent a "stimulation whereby all humoral and cellular factors of resistance are keyed to the very highest pitch." He believes that a general reaction must be produced in order to obtain any therapeutic effect.

A method by which the same sort of stimulation is obtained is that of treating the skin by light or by tuberculin applied by scarification, as in the Pondorff method, to produce a skin reaction. This is accompanied by leucocytosis and mobilization of enzymes from the skin such as occurs after a protein injection. The use of iodides, according to Jobling and Petersen, also acts in somewhat the same manner, producing a decrease in antiferment and an increase in cell permeability. A patient receiving iodides gives a remarkable reaction to skin tests, small amounts of tuberculin producing a suppurative lesion instead of the usual slight wheal.

Besides the general reaction produced by a protein injection, the focal reaction is also important. This is due to the same complicated reaction which occurs most markedly at the site of the lesion. Any cells which have previously reacted to an infection will respond for long periods to the injection of any protein. In this way Petersen would explain the lighting up

of a chronic cholecystitis or appendicitis during tonsillitis or sinusitis, considering this more probable than Rosenow's theory that bacteria from the tonsils or sinuses actually localize in the affected viscera—i. e., according to Petersen, the cholecystitis is a nonspecific focal reaction to absorption of bacterial protein from tissues.

The property of causing a general and focal reaction on injection into the body is common to all proteins. Almost every protein-containing substance one can think of, except ordinary street dirt, has been used for its nonspecific effect, Petersen listing over eighty such substances. This includes a number of protein split products, albumoses, deuteralbumoses and peptone, which produce the same phenomena in varying degree. While the proteins produce sensitization, the split products do not, but rather an increased tolerance to further doses.

This brings up the antigenic properties of the proteins, which are of especial importance to anyone who is treating asthma or hay fever. It is well known that any protein can produce specific antibodies and can sensitize the organism against it. Whether the whole protein molecule is necessary for this sensitization is still a matter of debate. Black, Grove and Coca have prepared pollen extracts which will sensitize animals and with which patients can be desensitized, and claim that they are protein free, since they are not affected by digestion with trypsin or by dialysis. The pneumococcus extract prepared by Avery was said to consist of glucosides. Kollmer thinks, however, that these workers have not excluded the possibility that minute amounts of protein are present in the extract, and believes the production of anaphylaxis by a non-protein substance is as yet unproved. Sensitization to certain drugs may be a slightly different phenomenon, which Kollmer prefers to call hypersensitiveness in distinction to anaphylaxis which is produced by proteins. He believes the site of the sensitization is in the cells, not in the blood stream, and that desensitization depends on exhausting the antibodies in the cells. McKenzie and Baldwin were able to desensitize the nasal mucosa of hay fever patients by local applications of pollen extracts. It must not be forgotten that besides pollen, horse dander and a few other causes of hay fever and asthma, bacterial proteins may play the same role, so that a sinusitis may

be the cause of sensitization and attacks of asthma as bacteria are subsequently absorbed or ingested.

Hay fever is often a cause of sinusitis through swelling of the nasal mucosa, and by sensitization to the bacteria a vicious circle is established to which Coates has called attention. Wilmer believes such sensitization is the principal cause of asthmatic attacks out of season in persons sensitive to pollen, and treats such patients with a mixed vaccine made from the nasal flora as well as with pollen. Using 215 different proteins, he found that 70 per cent of 100 cases of bronchial asthma gave positive reactions, usually to a relatively small number of proteins, horse dander, wheat, bacteria, pollens, cat hair and duck feathers.

The fact that hay fever and asthma undoubtedly show a distinctly hereditary influence is interesting, as according to our present views of heredity an acquired sensitivity can hardly be transmitted. Doerr has explained this as an inherited ability to produce antibody in response to a group of stimuli, while Kollmer believes it depends upon an inherited peculiarity of smooth muscle or of its sympathetic innervation.

A condition which has recently been brought into etiologic relationship with allergy is that of vernal conjunctivitis. Fort and Townsend, in 1924, both tested patients for sensitivity to the pollens and found that a number gave positive reactions and improved on desensitization, all but one of which were sensitive either to pollens or foods. One was sensitive to wheat and was relieved entirely when this was removed from the diet, and the same course was observed in another patient sensitive to egg-yolk. One patient sensitive only to spring pollens cleared up promptly on June 15. Three adults desensitized to pollens remained free from attacks for a year, with recurrence the following year.

H. Lagrange reports four cases sensitive to pollens. Both he and Lemoine also believed in a relation between endocrine disturbance and allergy, Lemoine using parathyroid and Lagrange orchitic extract in treatment, but since the mechanism of such a relation is unknown, the importance of either factor is hard to determine. A study of skin tests in these cases would certainly seem justified, especially in the more severe cases. In a few studied in Omaha, I have only had one clean-cut

result suggesting allergy as a cause, a child sensitive to short ragweed whose attacks occurred in August at the season of its pollenization.

Let me now consider some of the proteins of the eye which have a direct bearing on certain important problems. A good deal of study has recently been directed upon the vitreous in the light of modern chemistry. According to Matthews, the vitreous contains 0.12 to 0.19 per cent of protein, consisting of globulin, serum albumin and a mucoid substance differing in some properties from true mucin. This is embedded in a network of fibrils composed of collagen. The rest is water. Baurmann found that on dialyzing normal vitreous only 1/1000 of its weight was left, and that on adding water to this it swelled up to its former bulk. By ultramicroscopy he found a structure resembling that of certain soaps in the gel phase, fibrils 15 μ in breadth and up to 30 microns long being present everywhere, enclosing clear spaces. There was no evidence that these fibrils are cellular or the direct product of cell growth like the lens fibers. The vitreous cannot be considered a tissue, he believes, but is a specialized form of secretion, the fibrils representing the solid phase of a colloid and the water being bound to the small amount of protein by molecular attraction due to the immense surface of the protein particles. The ultramicroscopic size of the fibrils, approaching in size the wave lengths of light, accounts for its transparency. Baurmann found that on standing, the structure changed markedly, the fibrils becoming granular and disappearing as the vitreous liquefied. Redslob suggested that this tendency to liquefy on exposure to air, trauma or slight changes in reaction probably explains the liquefaction of vitreous in pathologic conditions. This French observer agreed that the vitreous is a gel, but found a more condensed substance around it which forms the hyaloid membrane, and a definite space running through it longitudinally which corresponds to the canal of Cloquet. Fischer first called attention to the swelling of vitreous in acid solutions and suggested that glaucoma might be the result of such swelling in acidosis. It is a clinical fact, however, that in the extreme acidosis of diabetic coma the eyes become very soft. Baurmann showed that the isoelectric point of the vitreous is PH 4.4, so that the degrees of acidosis compatible

with life would only bring its reaction nearer to this and cause shrinking. He believed it to be normally at a reaction corresponding to its maximal swelling, stability and transparency. Redslob, however, found that increasing the alkalinity caused further swelling up to P. H. 8.2, a definite amount of swelling following each change of 0.1 in the P. H. Since he found that .02 cc. of fluid added to the human eye at normal tension raised the tension to 60 mm. (Schiotz), he believes it is quite possible that changes in tissue alkalinity occurring during life might cause enough swelling of the vitreous to result in glaucoma.

Two proteins of the eye, that of the uveal pigment and of the crystalline lens, occupy a special place by their immunologic reactions, and much experimental work has been carried out with them. This special place is due to their production of organ-specific rather than species-specific antibodies when injected into animals. A rabbit injected with beef-lens shows precipitins and complement binding antibodies not only for beef-lens but for lens-proteins of all species and fails to show antibodies to beef-serum. This was first pointed out for lens protein by Uhlenhuth, whose observations were confirmed by Hektoen and others. Hektoen found that alpha and beta crystallin produced distinct precipitins which were organ and not species-specific. Elschnig and independently of him, Kummel, in 1910, investigated the protein of uveal pigment, and found that it also produced organ-specific antibodies. Upon this, as is well known, was founded Elschnig's anaphylactic theory of sympathetic ophthalmia, according to which the uveal pigment, set free after a penetrating wound with resulting inflammation, sensitizes the body, so that when further uveal pigment is set free an anaphylactic reaction results in the second eye where such pigment is present. Experimental work supporting this theory was brought forward in this country in 1918 and 1921 by Alan Woods, who in subsequent papers has made the theory quite familiar to American ophthalmologists. This is not the place to add to the already voluminous polemic on the subject, and I have elsewhere summarized a number of reasons for considering the anaphylactic theory as yet unproven and for believing that some organism or organisms, or a filterable virus, will be found to be the cause of sympathetic ophthalmia. Woods has used a uveal pigment extract

for the early diagnosis of sympathetic ophthalmia and for its treatment. He reports that cases of sympathetic ophthalmia given a negative complement fixation but positive skin test with pigment. He has treated ten cases with desensitizing doses and reports result indicating a definite therapeutic effect, especially in two patients on whom subsequent cataract operations were performed with very little reaction. Fodor, in 100 cases of various kinds, obtained conflicting results which caused him to reject the skin tests as a means of deciding for or against enucleation of a suspected eye. In two cases of sympathetic ophthalmia I obtained negative skin tests with Woods' antigen. The value of a skin test with pigment, if it proves to be a means of early diagnosis in sympathetic ophthalmia, would be so tremendous that it is hoped other observers will give the method a thorough trial and report their results.

Uhlenhuth's description of the organ-specificity of lens proteins has given rise to a great deal of experimental work of varying degrees of importance. Great scientific interest was aroused by the reports of Guyer and Smith that they had succeeded in producing cataract and other serious eye defects in offspring of rabbits treated with the serum of chickens immunized to lens protein. This work, which would necessitate a modification of our ideas of heredity, has apparently found widespread acceptance, and the work of Huxley and Carr-Saunders, of Finlay, and of Poynter and Allen, who repeated his experiments without once producing such effects, has not overcome the impression created by Guyer's work. Von Szily and others had shown that defects exactly like those shown by Guyer existed quite frequently among rabbits as a result of faulty closure of the fetal cleft and were transmitted to their offspring. Such a family defect occurred among the rabbits which Poynter used as controls in his experiments, which had received no treatment. Thus it seems as if we would not be forced to change our ideas of the nontransmissibility of acquired characters on the basis of Guyer's work.

Verhoeff and Lemoine, in 1922, attempted to explain reactions following cataract operations with retained cortex as due to a hypersensitivity to lens protein, the cases showing positive skin tests with a lens protein emulsion. About 8 per cent of normal persons showed this hypersensitivity, and these

persons, they believe, are apt to develop reactions if cortex is left in the eye. Steinberg and I also found a certain number of persons who gave positive skin tests, and one case especially which showed a severe ocular reaction to retained cortex with marked sensitivity to lens protein as shown by skin tests. A number of cases, however, with ocular reactions gave negative skin tests, so that we concluded that some other factor than hypersensitivity must be present to account for the majority of ocular reactions. Certain evidence seemed to indicate that split products of the lens were themselves toxic. Von Rötth was unable to obtain any positive skin tests and rejects the possibility that ocular reactions result from hypersensitivity.

Courtney has recently claimed that the skin test with lens protein becomes positive after cataract operation when cortex is left in the eye. In several hundred skin tests I have never seen this occur and doubt if enough lens protein can be absorbed in this way to produce sensitization. Out of a recent series of fifty skin tests on cataract patients, using a filtrate of lens protein prepared by Woods' technic, only two were positive. Several cases showed marked ocular reactions with retained cortex were retested and gave negative results. My observations would not lead me to place very much value on the clinical use of skin tests as a guide to the type of operation to be performed, as is claimed by Verhoeff and Lemoine. Further observation of such tests, however, will be interesting and it is to be hoped a number of observers will report their results.

Because it has aroused some interest, even among ophthalmologists, the work of Davis should be mentioned. Davis reported, in 1922, the results of injecting an extract of beef lens subcutaneously for the treatment of cataract. He believed that such injections stimulated the production of cytolytins which would dissolve the cataractous material. Why, if effective, it would not also dissolve the normal lens fibers, producing aphakia, is not explained. Davis seems to have been unaware of the previous work of Römer, who, working on a different theory, used a lens extract which he called lentokalin, by injection and by mouth, for the treatment of cataract. A good deal of work was done with this and other allied preparations.

but the results of all this work, as recently summarized by Siegrist, must be considered doubtful, and from a scientific standpoint negative. Ellis recently compared a series of cases treated by Davis' extract with a series of controls without noting any important differences. Not only is there no sufficient theoretical or clinical evidence for encouraging the trial of lens extract in the treatment of cataract, but the possibility must be considered that one may sensitize a patient by such injections so that if operation later becomes necessary it may be attended with a severe ocular reaction in Verhoeff's sense.

More important than any of this work, to my mind, is the study of the chemistry of the lens in its relation to the possible cause of senile cataract. This study, the foundations for which were laid in 1894, by Moerner, has been continued by Jess and Goldschmidt in Germany, Dorothy Adams in England, Woods and Burky in this country and others. A recent valuable summary of the literature was contributed by Tassman (*Archives of Ophthalmology*, 1928). While it cannot be said that a satisfactory explanation of senile cataract has resulted from this work, it has at least revealed certain actual phenomena which take place in the cataractous lens, and I believe it is by further study in this field that we may arrive at an understanding of cataract. Moerner showed that the lens contained 35 per cent of protein, the rest being water with a small amount of salts and fatty bodies. The insoluble protein, called albumoid, made up 15 per cent of the lens mass, and the soluble protein 18 per cent, of which 6.8 per cent was alpha crystallin and 11 per cent beta crystallin. The .2 per cent of albumin found by Moerner is probably identical with the gamma crystallin recently described by Woods. Jess found that the chemical composition changed with age. The weight of the lens increased, due chiefly to an increase in protein, and chiefly in the insoluble albumoid, which in cows from 1 to 16 years of age increased from 5.6 per cent to 21.4 per cent of the lens weight while the soluble proteins decreased from 25 to 15 per cent. In senile cataract, instead of the normal increase in weight, he found a constant decrease, both in water and protein. Of the latter, the soluble portion decreased markedly, while the insoluble portion remained about the same. Jess considered that in the senile lens the crystallins were

changed to albumoid, while in cataract formation the crystallins, especially beta crystallin, may break down and dialyze out of the lens.

This loss of the soluble proteins, and especially of beta crystallin, was held to be important, since beta crystallin is the chief repository of the aminoacid cystein. It was shown by Hopkins that tissue oxidation depends upon the presence of a combination of cystein and glutamic acid, which he called glutathione. This auto-oxidative mechanism must be considered especially important in the lens, which has no blood supply. It seems likely that the increase in insoluble proteins, with a loss in soluble protein bearing the oxidation-reduction mechanism of the lens are important factors in cataract production, perhaps the most important factors. As to the cause of these changes themselves, we are still in the dark.

If any practical conclusions can be drawn from this mass of theory, they would be the following:

1. Any protein on injection is capable of producing a general and focal reaction, which depends on blood changes, mobilization of enzymes which break down toxic proteins and increased permeability of cell membranes.
2. A number of other therapeutic agents, such as potassium iodide, probably depend on similar phenomena.
3. It is doubtful if any nonprotein substances can produce true anaphylaxis.
4. The vitreous is a colloidal substance, whose iso-electric point is at a higher acidity than is found in the body. Hence acidosis must produce a shrinkage of the vitreous, and alkalosis may produce a swelling sufficient to cause glaucoma.
5. The organ-specificity of the uveal pigment is probably not absolute, the evidence in favor of an anaphylactic theory of sympathetic ophthalmia is not complete and the value of the skin tests with pigment, although worthy of further investigation, has not been proven.
6. Ocular reactions as a result of hypersensitivity to lens protein probably occur, but the majority of reactions following cataract operation seem not to be of this nature.
7. There is no theoretical or clinical evidence to justify the use of lens extracts in the treatment of cataract.

8. The increase of insoluble protein and loss of reducing power in the senile and cataractous lens are important factors in the etiology of cataract.

BIBLIOGRAPHY.

- Guyer and Smith, Poynter and Allen, etc., quoted by Duke-Elder: Recent Advances in Ophthalmology, Philadelphia, 1927, pp. 77 and 233.
Davis: Am. International Congress of Oph., Washington, 1912, p. 284.
Siegrist: Der Graue Alterstar. Berlin, 1928.
Ellis: Arch. of Oph., 1928, 57, p. 46.
Tassman: Arch. of Oph., 1928, 57, p. 361.
Reis: A. J. O., 1912, 80, p. 588.
Jess: A. J. O., 1912, 71, p. 259. Zeit. f. Biol., 1913, 61, p. 93. A. J. O., 1921, 105, p. 428. A. J. O., 1921, 109, p. 453. Zeit. F. A., 1921, 46, p. 237.
Goldschmidt: A. J. O., 1917, 93, p. 447. A. J. O., 1924, 113, p. 160. K. M. F. A., 1924, 72, p. 777.
Adams: Proc. Royal Soc. Med., Series B, 1925, 98, p. 244. B. U. O., 1925, 9, p. 281.
Woods: Arch. of Oph., 1918, 47, p. 161. Trans. Oph. Sec. A. M. A., 1921, p. 105. Arch. of Oph., 1928.
Lemoine: Tr. Am. Acad. Oph. and Otolaryng., 1925, p. 198. Arch. of Oph., 1929, 1, p. 706.
Lagrange, H.: Presse Med., 1923, 31, pp. 112 and 380.
Verhoeff and Lemoine: Int. Cong. of Oph., 1922, p. 234. A. J. O., 1922, 5, p. 700.
Lemoine and MacDonald: Arch. of Oph., 1924, 53, p. 101.
Gifford and Steinberg: J. A. M. A., 1925, 85, p. 351.
Courtney: A. J. O., 1929, 12, p. 20.

NASOPHARYNGEAL ABSCESS: A REPORT OF
ONE HUNDRED FIFTY-FIVE CASES.*

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The lymphoid tissue in the nasopharynx has a definite anatomic arrangement within the structure of the normal adenoid. This anatomic arrangement is maintained when the adenoid persists into adult life and can usually be recognized even when the adenoid structure has become distorted by hypertrophy.

Normally, the adenoid consists of sheets or combs of lymphoid tissue, separated by clefts, and attached to the vault of the nasopharynx along anteroposterior lines. The central cleft begins behind the attachment of the vomer, where it is shallow, and extends backwards in the median line, becoming deeper at its posterior extremity, where it ends in a fossa known as the recessus medius. On each side of the central cleft is a central comb. Outside of each central comb there is a first lateral comb, separated from the central comb by the first lateral cleft, and usually there is an incomplete second lateral comb separated from the first by the second lateral cleft. There are, therefore, a central and two lateral clefts, running parallel to each other in an anteroposterior direction. These clefts unite posteriorly in the recessus medius. Frequently the second lateral combs come together posteriorly behind the recessus medius, forming a transverse comb.

In the embryo, the hypophysis cerebri takes its origin from an indentation in the nasopharynx, the location of which is identical with the recessus medius of the adenoid. The indentation grows upward into a prolonged stalk terminating in a mass which becomes the anterior portion of the hypophysis.

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The stalk itself atrophies but, as in all embryonal structures of this type, remnants of the lumen occasionally remain. Such a remnant is in very rare instances found in the nasopharynx. It consists of a cyst located behind or above the recessus medius. This is the so-called pharyngeal bursa. It is rarely found, but when present may become the seat of a suppurative process.

In 1885, Tornwaldt, of Danzig, published in the form of a monograph, a series of clinical observations in which he declared that he had seen numerous cases of abscess formation in the region of the recessus medius. These abscesses varied in size from one or two minims to several centimeters, and the contents varied from serum or clear mucus to creamy pus. Because of the fact that these abscesses are situated in the place where the pharyngeal bursa originated, Tornwaldt concluded that these cases were all cases of pharyngeal bursitis.

His observations were not generally confirmed; first, because others did not see such cases nearly as frequently as Tornwaldt did, and secondly, because the presence of an uninfamed pharyngeal bursa was a most rare anatomic find. For a number of years an animated discussion followed the announcement of Tornwaldt. In a general way, the outcome of the discussion was that because the pharyngeal bursa is a rare anatomic find, therefore, the cases which Tornwaldt saw must be very unusual cases, a conclusion which seemed to be verified by the fact that no others were able to report similar cases, in like number.

This conclusion was erroneous, for two reasons: First, because Tornwaldt's claim that these abscesses were due to inflammation of the pharyngeal bursa was unequivocally accepted, and secondly, because the negative findings of others were given equal weight with the positive findings of Tornwaldt. A study of the clinical reports of Tornwaldt's cases has convinced me that the cases which he saw were identical with the cases upon which this report is based, and which, as I will try to prove, can be attributed to another origin than inflammation of the pharyngeal bursa, namely, to an inflammation of the recessus medius of the adenoid. The frequency with which Tornwaldt saw these cases was due to unusual diligence and skill on his part, for he made it a routine prac-

tice in his clinical work to cocaine each one of his patients sufficiently to enable him to introduce a self-retaining palate retractor so as to make possible the examination of the nasopharynx in a convenient and thorough manner.

In the year 1910, the writer devised a direct speculum for the examination of the nasopharynx. With this instrument it is possible with a minimum amount of cocaine, and with a minimum amount of circumstance, to make a thorough examination of the vault of the nasopharynx. Because the use of this instrument has become a matter of routine practice in my office I have been able to verify the observations of Tornwaldt not only as to the presence of these abscesses in the nasopharynx but also as to their frequency; but my observations have convinced me that these abscesses are due to suppuration in the remnants of previously inflamed adenoid tissue and not to inflammation of a supposed pharyngeal bursa. This conclusion is based on the following data:

1. A study of the ages of the patients presenting these abscesses shows that the largest number of cases occurred between the ages of 20 and 30—72 of the 155 cases. The next most frequent decade was from 30 to 40—32 cases. During this period of life, from the 20th to the 40th year, which is the period immediately following the involution of the adenoid mass, two-thirds of the cases occurred.

2. The statistics show that of the 155 cases, only 11 patients stated that they had at any time been operated upon for the removal of adenoids. Ninety-three per cent of the patients, therefore, never had their adenoids removed.

3. If the nasopharynx of adults whose adenoids have not been removed by operation be studied, there will frequently be found in the mucous membrane of the nasopharynx one or more openings into which a probe may be introduced for a distance of $\frac{1}{2}$ cm. Most frequently a single opening is found occupying the site of the recessus medius of the adenoid. When an additional opening is found, it is almost invariably situated in the median line anterior to the above mentioned opening. Sometimes a series of three or four successive openings are found in the median line, and the probe shows that they communicate with each other. The bridges of tissue between the openings are fragile and can easily be broken down

with a probe, converting the series of openings into a slit corresponding to the median cleft of the adenoid.

4. Where an abscess is present, a small dimple can sometimes be seen on the surface of the swelling. When the abscess is opened with the probe, the opening is converted into a slit. If nothing further is done the parts will soon present exactly the same appearance which is seen in a nasopharynx which presents one of these cryptlike openings but which has never been the seat of an abscess.

5. I have actually observed cases passing from the stage of abscess to the stage of a crypt and reappearing again as an abscess.

6. With one or two exceptions all these abscesses occurred in the median line.

7. It is therefore evident that this condition of abscess formation is one which occurs in adults whose adenoids have never been removed by operation. It occurs shortly after the period when the involution of the adenoid mass has been completed, and in cases in which adhesions between the combs of the adenoid tissue have converted the median cleft into a more or less completely closed cavity.

The history of these cases and the appearance of the nasopharynx corresponds closely with many of the histories and descriptions recorded by Tornwaldt, and I am convinced that the cases that I have seen are the same as those which he described but which he erroneously attributed to an inflammatory process in a supposedly persistent pharyngeal bursa. On the other hand, I have seen one or two cases in which a larger cavity appeared which could not be entered with the probe but required a knife, which contained a larger amount of serous fluid and which were considered to be true cases of pharyngeal bursitis.

DIAGNOSIS.

A study of the statistics of this series of cases shows that there is a preponderance of females, there being 90 females and 65 males.

The ages vary from 5 years to 65 years, with most of the cases occurring between the ages of 20 and 40.

It is of some interest to note that the majority of the cases occurred either in the late spring, from March to June inclusive, or in the late fall, from October to January inclusive. This seasonal distribution is probably associated with weather conditions and might be expected to vary with the climate.

It is also of interest to note the frequency with which these cases occurred in relation to other nasal and nasopharyngeal conditions. The total number of cases was collected over a period of years. But of 760 patients of all kinds of nose, throat, laryngeal and endobronchial diseases seen in private practice during the year 1928, there were 28 cases, or 3.8 per cent, with abscesses of this kind. During the first four months of this year, of 315 patients of all kinds, there were 13 nasopharyngeal abscesses, or 4.1 per cent. This slight increase in the present year is due to the fact that it includes the months in which the disease is most common and that during this period there was an epidemic of upper respiratory infections.

The local diagnosis depends upon the use of the direct nasopharyngeal speculum. When a reflecting instrument is used to examine the nasopharynx the light rays are projected upon the pharyngeal mucosa from a reflecting surface which is placed far back near the posterior pharyngeal wall. The angle of incidence of the rays as they strike the surface of the nasopharyngeal mucous membrane is greater than the angle of total reflection of the mucus which covers the surface. In like manner, any light rays which may emerge from the surface of the mucous membrane are totally reflected at the surface of the superficial layer of mucus. Therefore, unless the nasopharynx has been entirely freed of mucus and maintained free of mucus during the examination, all that will be seen in a reflecting instrument will be a uniform pinkish sheen with the details of the texture of the surface obliterated. In the majority of cases it is practically impossible to recognize the contour of the nasopharynx, which is essential for the diagnosis of the condition. On the other hand, with a direct speculum the presence of a small quantity of mucus does not interfere with the visibility of the surface of the mucous membrane and, besides, the surface can easily be kept free of mucus with suction.

An abscess is characterized by the presence of a swelling in the region of the recessus medius, but the swelling may not manifest itself as a rounded protuberance but merely as an elevation of the concavity of the vault of the nasopharynx, a flattening out of the concave surface. When the swelling can be recognized, it may be red, smooth and glistening or it may present a dull, grayish, almost polypoid appearance. Occasionally a dimple is found on the surface of the swelling. Not every such swelling, however, contains pus, and the diagnosis is not complete until the swelling has been opened and its contents noted. In all cases, except one or two, it has been possible to penetrate the swelling with a probe, whereupon pus was found to exude alongside of the probe. The puncture wound made by the probe may then be converted into a slit and the contents of the cavity completely evacuated. It was in this way that each one of the 155 cases upon which the present article is based was diagnosed, and it was only after the fluid contents of the cavity were actually seen that the diagnosis was considered complete. Of the 155 cases, the character of the contents was noted in 142. In 121, or 86 per cent of these, the contents were recorded as thick, creamy pus. In five of these, exuberant granulations could be seen protruding from the wound after the pus was evacuated. In the remaining 21 cases, or 14 per cent, clear, thick, glairy mucus was evacuated.

Unless and until the rhinologist acquires a familiarity with the appearance of the nasopharynx in the direct nasopharyngeal speculum, and as long as he depends entirely upon reflecting instruments for the examination of these parts, it will be impossible for him to recognize the presence of these abscesses in all of the cases, and it will be impossible for him to treat them in an efficient, surgical manner.

In practically all of the patients the abscess in the nasopharynx was not the only pathologic condition present, nor was it seemingly the most important. The associated conditions of the nose and throat include practically all of the diseases generally found there. In over one-third of the cases diseased tonsils were associated with the nasopharyngeal condition, and deviated septa and accessory sinus disease were not uncommon.

A few cases of posterior cervical adenitis seemed to be directly associated with the presence of these abscesses.

As already indicated above, the abscesses had a tendency to open spontaneously and discharge their contents, and then to close without indicating the fact by any evidence of an acute process. Under these circumstances, it is manifestly impossible to distinguish the symptoms caused by the presence of such a small abscess from those caused by the associated diseases. Nevertheless, by reviewing the figures obtained from a study of these 155 cases it was possible to obtain some significant data.

For this purpose, the symptoms presented by the patients were separated into groups. The first group are those symptoms which might reasonably be attributed to the presence of an open abscess cavity discharging its contents into the nasopharynx. This group included such symptoms as nasal discharge, postnasal discharge, and frequent colds, including hoarseness, foul taste or odor, nausea, hawking and coughing, attacks of sneezing. The total number of patients presenting these symptoms was 103, or 66½ per cent.

The second group of symptoms are those which might reasonably be attributed to a closed abscess cavity in the nasopharynx. This group includes headache and pains in various parts of the head and neck, including a number of cases diagnosed as sphenopalatine neuralgia, nasal obstruction, sore throat, nasal speech, cervical adenitis. The number of cases in this group was 108, or 68 per cent. It is therefore evident that about two-thirds of the patients complained of symptoms attributable to an open abscess in the nasopharynx, and about two-thirds of the patients complained of symptoms due to a closed abscess. These figures can only be reconciled by assuming that approximately one-half of all the patients complained of symptoms when the cavity was closed as well as of symptoms when the cavity was open; that of the remainder, some complained of symptoms only when the cavity was open, and some only when the cavity was closed.

The headaches of which the patients complained included all kinds of pain in the head and neck, but a number of patients, perhaps one-half of those complaining of headache, de-

scribed the headache in a peculiar way. The headache is felt at the back of the head and resembles the pain of sphenoid sinusitis. However, when a patient with sphenoid sinusitis is asked to point out where his headache is situated, he will place his entire hand upon his occiput; but when a patient who has a headache due to one of these abscesses tries to point out its location he will place one finger upon the nape of the neck, just below the occipital bone—a point which corresponds closely to the location of the abscess. In other words, the headache due to a nasopharyngeal abscess seems to be much more localized than the headache of sphenoid sinusitis. Although this particular type of headache occurred in only a minority of all the patients, when it did occur an abscess was invariably found in the nasopharynx, and the opening of the abscess was invariably followed by the disappearance of the headache. In some instances, these headaches were severe and many unsuccessful attempts to relieve them had been made. It is the only symptom of these abscesses which seems to me to be so definitely and clearly associated with the presence of the abscess that it could be regarded as pathognomonic.

The next group of symptoms are those relating to the ear, and it is very surprising that in a suppurative disease of the nasopharynx there were only 23, or about 15 per cent, of the patients who complained of any ear symptoms whatsoever. Only one-third of these, or 5 per cent, at any time had a suppurative otitis, either acute or chronic. The other ear symptoms were vertigo, tinnitus, earache and deafness.

In like manner, it is perhaps equally surprising that there were comparatively few, not more than 5 per cent of the total, who complained of focal symptoms, such as nephritis, rheumatism, persistent fever, etc. Nevertheless, in some of these cases the relief of rheumatism followed as promptly as it sometimes does after the removal of tonsils, and there were three cases of persistent slight rise of temperature which were relieved by the removal of such abscesses.

There were a large number of nervous symptoms whose relation to the abscesses may be obscure; they included neurasthenia, insomnia, reflexes of various kinds, and aches and pains in various parts of the body. One case of frequent

micturition was immediately relieved after opening such an abscess.

A number of instances occurred in which a diagnosis of accessory sinus disease had been made and for which various sinuses had been operated upon. In spite of the fact that the intranasal appearances seemed to indicate that the sinuses were healed, nasal discharge and headache persisted and were relieved only when the abscess in the nasopharynx was opened.

The number of cases seen in hospital and dispensary practice was very much smaller than those seen in the writer's private practice, but this fact itself is significant. It is probably due to the circumstance that the writer's practice is entirely a consultant practice and that the patients that are brought to him are those in whom careful diagnosis and skillful treatment failed to relieve the patients. In other words, it is among the unsuccessful cases of the average rhinologist that these cases have been found. This fact should make the recognition and treatment of these nasopharyngeal abscesses a matter of great interest and importance to all rhinologists, for in the writer's practice one of these abscesses was found in every twenty-five patients. It is certain that a pathologic condition which occurs in the practice of a single rhinologist every ten days cannot be dismissed as a rare or unusual or unimportant disease. If this figure truly represents the proportion of undiscovered nasopharyngeal abscesses among the unsuccessful cases of other rhinologists, a better knowledge of the diagnosis and treatment of this condition will add a measurable degree to the therapeutic efficiency, and therefore to the professional prestige of our specialty.

The treatment of these cases, as has already been suggested above, is carried out through the direct nasopharyngeal speculum. The cocainization necessary for the introduction of this instrument is as follows:

Two applicators are wound with absorbent cotton for a distance of $1\frac{1}{2}$ cm. from the end; the tuft of cotton should not be more than 4 mm. in diameter and the end rounded off smoothly. About 2 cm. of the end of the applicator are bent to a shape corresponding to the shape of a eustachian catheter. These applicators are saturated (but not dripping) with

from 5 to 20 per cent cocain solution with adrenalin and introduced through the nostril with the bent part downward so that the bent part comes to lie on the upper surface of the soft palate. They are left in situ for ten minutes. The instrument is introduced in the usual manner until the swelling in the nasopharynx is brought into its lumen. The use of suction may be necessary to keep the site clear of secretion, especially if there is nasal discharge from an associated sinusitis. A stiff steel probe is applied to the swelling. It can easily be thrust through the walls of the abscess cavity. A small quantity of secretion usually exudes alongside of the probe. The probe is then forcibly moved downwards and backwards, converting the puncture wound into a slit and evacuating the contents of the cavity. There is usually brisk but temporary hemorrhage. When this has subsided an application of 20 per cent solution of nitrate of silver may be made to the interior of the cavity. In many cases this is sufficient to eliminate the disease. The lateral walls of the cavity separate, the recessus medius becomes shallow and the surface of the nasopharynx smooth. In a number of instances, however, the cavity reforms. This is due to the fact that the walls of the cavity, which are composed of the central combs of the adenoid, still contain a considerable amount of lymphoid tissue. They remain thick and rounded and maintain the original formation of the central cleft and recessus medius. The removal of this remaining lymphoid tissue is therefore indicated. This can be accomplished without difficulty through the direct speculum, using for this purpose an ordinary punch forceps of sufficient length to reach through the speculum. Occasionally the amount of lymphoid tissue is unexpectedly large and a more complete operative procedure is necessary. The removal of this tissue then becomes equivalent to the removal of adenoids from the nasopharynx of the adult, and may be accomplished by a procedure which serves this purpose. The writer has successfully used the method taught by Dr. Joseph C. Beck, which consists of introducing rubber cords through the nares, bringing them out of the mouth and using them as palate retractors, and in this way curetting the nasopharynx under direct vision.

Not the least important feature in connection with these abscesses is the attitude which rhinologists have assumed

towards this condition. Although at one time it was the subject of animated discussion under the name Tornwaldt's disease, in late years it seems to have been omitted almost entirely from consideration and to have become lost to rhinologists as a factor of any importance in the pathology of the nose and throat. Not only do we find discussion of the disease almost entirely absent from the literature, but of the textbooks published in the English language during the last ten years half describe, under the name of Tornwaldt's disease, an inflammation of the pharyngeal bursa. Inflammation of the pharyngeal bursa is an entirely different pathologic condition, as the bursa is situated behind the mucous membrane of the nasopharynx and behind and above the recessus medius. The contents of the pharyngeal bursa are more often clear serous fluid or glary mucus. When the bursa is the seat of suppurative inflammation, the differential diagnosis from suppuration in the recessus medius is difficult, but the writer believes that a distinction can be made by the surgical procedure necessary for the opening of the abscess. The recessus medius being situated entirely in the remnant of adenoid tissue, the abscess cavity can be entered easily by means of a probe and the cavity opened in this way. It is practically never necessary to use a knife. On the other hand, when the cavity to be opened is a pharyngeal bursa, the probe cannot be made to enter the cavity. The mucous membrane of the nasopharynx is too resistant for this simple procedure. It becomes necessary to use the knife.

In those textbooks which have described Tornwaldt's disease it is always pharyngeal bursitis which is described. I have found only one exception to this rule. In the first edition of Ballenger's "Textbook on Diseases of the Nose and Throat," Tornwaldt's disease is described definitely as an inflammation of the recessus medius. In the year 1912, a book on "Diseases of the Nasopharynx," by Adair Dighton, was published in England. Dighton accepts the description of Tornwaldt's disease published by Ballenger and repeats the description almost verbatim. The same description is repeated in the later editions of Ballenger. With this exception, there is no publication that I have seen in which Tornwaldt's disease is described as an inflammation of the recessus medius. I have found no

other textbook in which inflammation of the recessus medius as such is described at all. The disease has practically been lost to rhinologists, and the only excuse which the writer has for attempting to resurrect this lost disease and to save it from oblivion is the fact that never a fortnight passes in which he does not see at least one case and that the relief of a number of obstinate cases which has been accomplished by the discovery and cure of these abscesses have been among the most satisfactory cases in the writer's experience.

XXXIV.

NONINFECTIOUS FACTORS IN THE ETIOLOGY OF SINUS DISEASE (WITH SPECIAL REFERENCE TO THE RETICULOENDOTHELIAL SYSTEM).*

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Dissatisfaction with surgical operations for the relief of sinus disease has become a commonplace among patients, and rhinologists are not infrequently asked to devise palliative treatment for persons who refuse operative procedures. Reproach is made by some of our members that we have progressed little beyond the stage of merely "opening and draining" these cavities. Others are reviving and improving the most radical procedures, sacrificing perhaps less turbinal tissue but removing the lining of every sinus as completely as possible.

Between these extremes—nihilism and radicalism—stand out the researches of Dean, Shurly and others upon the effects of malnutrition and similar factors. We have witnessed the massacre of turbinates, tonsils, teeth and sinuses by clinicians schooled in facile technic, eager for quick results, yet ignorant of basic physiology and pathology. Diagnosticians demanding the elimination of alleged infective foci may be held in some measure responsible for this situation. As Draper says, "The habit of clinging too eagerly to mechanistic tradition may constitute a danger which works against enterprise."

Perhaps it may be salutary to discuss other factors in sinus disease, omitting infective agents altogether.

Among children in the same household, with no differences in environment, diet or exposure, one will develop sinus dis-

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ease, the others not. Adults in offices, college students, industrial workers, swimmers using common facilities—countless groups of human beings exposed to equal hazards daily remain free from sinus involvements, save for a few susceptible mortals in each group.

That infection is not alone or mainly to blame has recently been demonstrated by Ashley and Frick, working under the direction of Dr. H. J. Sears, professor of bacteriology, in our department of the University of Oregon Medical School, during the past year. Using the most careful technic, out of a large number of children with positive nasal symptoms, "dark" antra, nasal discharge and purulent fluid secured by needling, they found a considerable number negative on culture by various methods. Also they demonstrated, instead of the expected streptococcus, the hemolytic staphylococcus, the pneumococcus and similar virulent types, that fifteen different sorts of organisms were present, each in pure culture or combined with a single other germ. Many of these were ordinarily of feeble virulence; we have therefore repeated proof that certain factors have permitted organisms frequently harmless to acquire increased virulence and invade the sinuses of these children.

Among these extrinsic factors are those of environment: climate, including heat and cold, amount of humidity in the air. House heating systems which lack proper means for humidification are especially bad influences in this regard. Irritant vapors or gases—fuels, exhausts, industrial products—may cause decided lowering of local resistance, as also excessive smoking.

Physical factors external to the sinuses include, of course, direct trauma to the face—dental fractures, motor accidents, gunshot wounds and the like, in which the sinus lumen may fill up with serum or clots.

Influences of a general order which definitely lower resistance in susceptible persons include prolonged exposure, especially with chilling of the body surface; physical or mental overwork; repeated loss of sleep; dieting or starvation. If these factors be complicated by glandular wastage, as in severe emotional shock, excessive venery, thyrotoxicosis, resistance falls to a very low ebb.

Intrinsic factors predisposing to sinus disease have long had careful study. Anatomic factors—narrow, twisted, obstructed and deformed noses—may be hereditary or acquired. Allergic predispositions, often familial, may readily be excited to a point where new sensitizations become active, with super-added liability to stasis and infection. These may enter by inhalation, by skin contact, by the digestive tract, and may even be excited therapeutically, as by silver protein solutions. Local irritative phenomena may arise, even in nonsensitized people, from inhaled dust—cedar sawdust, cotton fiber, marble dust, coal, flour, metal filings, abrasives—usually an occupational exposure.

Accidental introduction of water into the sinuses during swimming or diving, mentioned many years ago by Luc, arrests ciliary action, washes away the defensive mucous film, and by this hypotonicity leads to edema of the sinus mucosa. These factors are independent of infection, which may or may not be carried in at the same time.

Metabolic disturbances, due, in the matured opinion of Dean, Shurly, Stucky, Barlow and others, mainly to vitamin A deficiency, are among the most important factors in the etiology of sinus disease. Further studies in dietetics and nutrition are in constant progress and bid fair to solve many of these difficulties, especially in childhood and adolescence.

Severe disturbances in the respiratory system may, on account of increased respiratory efforts, not only cause direct infectious complications in the sinuses, but also may interfere indirectly by causing turbinal hypertrophy and consequent stasis. This indirect effect merges, of course, with those little understood phenomena of turgescence, transudation and altered secretion which depend apparently upon derangements of the vegetative nervous system. Herein may be grouped not only a certain number of asthma cases, but also others of rhinorrhea, of periodic turgescence prior to and associated with menstrual disturbance, of sphenopalatine pain associated with swellings at the tuberculum septi, and the like. Such troubles are often intimately linked with dysfunction of the chromaffin-adrenal system and of the so-called glands of internal secretion; and undoubtedly many such cases are helped by appropriate though minimal dosage of the glandular sub-

stance—pituitary, ovarian, thyroid, parathyroid—which seems to be lacking. Such therapy is, however, unsually but a makeshift, and its failure discloses our ignorance of definite methods for influencing the autonomic nervous system.

Müller and other recent investigators make the claim that endothelial permeability to leucocytes, as well as their distribution, is regulated in large measure by autonomic nerve impulses. If vasoconstriction (sympathetic) be excessive, local leucopenia will be observed, while overbalance of vasodilator (parasympathetic) elements will cause local leucocytosis.

In a recent histologic study of sphenoid and ethmoid tissues removed at operation, MacMahon noted great thickening of the blood vessel walls, with connective tissue hyperplasia—a most constant and significant finding. Because of its meager physiologic function as part of a vestigial and disappearing group of cavities, the vascularity of sinus mucoperiosteum is far poorer than that of mucosa exposed directly to respiration; but its connective tissue elements are notably active in response to inflammatory stimuli.

Connective tissue, the so-called mesenchyme, is everywhere present throughout the body, and has, since Metschnikoff, been known to play a leading part in the body's reactions of defense. In our insistance upon purely infective phenomena, we have inclined too much toward consideration of the germ and how to kill it; perhaps too little toward study of defensive mechanisms.

The reticuloendothelial system, consisting not only of large cells of the splenic pulp and lymph nodes, Kupffer's stellate cells in the liver, neuroglia and nerve cells, but also of mesenchymal connective tissue elements in the adventitia of blood vessels, was traced by Marchand, Borel and Maximow, Ribbert and others, by vital staining methods, prepared the way for Aschoff and Kiyono's monumental study (1913) of the mesenchymal, mononuclear phagocytes of the connective tissues. Their findings have been confirmed and extended by Maximow, Jaffé, Cannon, Opie and many others.

Roughly stated, it is now accepted that vascular endothelium—not only special cells in distant structures like the bone marrow or spleen—is a primary factor in supplying the so-called histiocytes, which are most important in phagocytosis,

in the removal of débris, and in the formation of granulation tissue. Not only from the vessel walls and by filtration from the blood stream, but also from among the fixed cells of ordinary connective tissue, these cells originate. In the cellular syncytium are found undifferentiated cells with pale small nuclei, lying among the fibrils. From such undifferentiated cells, with unimpaired embryonal mesenchymal potencies, in connective tissue throughout the body in varying proportions, as well as from similar elements in the reticulum of the blood forming organs, are developed the small round cells which appear to combat inflammation.

Antibody formation is thought to take place mostly in the cells of the reticuloendothelial system. This would account for the demonstrated phagocytosis of bacteria placed on the skin and other epithelial surfaces. Conversely, exhaustion of the mesenchymal defense reaction accounts for the "agranulocytic" inflammations with spreading necrosis and rapid death, and for fulminant influenzal complications in which no impulse toward formation of a protective barrier of leucocytes exists.

Metchnikow of the Pasteur Institute recently divided immunities into two groups: First, a progressive adaptation to toxins or to unfavorable environment, in which our cells gradually *lose* their sensitiveness to the unfavorable agent; and, second, an immunity of defense due to *increased* sensitiveness of the cells—a heightened reaction to germs and parasites.

Defensive immunity is, then, a mobilization and sensitization of all cells and tissues, but most notably of the ever-present reticuloendothelial components of the ever-present connective tissue.

Sterile inflammations have been produced by irritative agents and have been found to increase resistance to the penetration of bacteria later introduced. Polymorphonuclear cells appear first, and later, when the tendency toward recovery is established, mononuclear cells predominate.

Intravenous injection of vital stains like trypan blue, or of pigments like colloidal silver, saccharated iron and india ink, produces a so-called "blocking" of the reticuloendothelial cells. The amount of vital staining—especially notable in the spleen and liver—is an accepted index of the functional activity of these cells. Accurate study has been made of the stimulation

by this "blocking" of the release of antibodies into the circulation. Small amounts of the "blocking" or irritant substance intravenously stimulate antibody liberation, while large amounts inhibit such liberation. Increased vital staining (functional activity) is also reported after weak X-raying of the spleen.

Wherein are these recent developments of interest to us?

We have got about as far as we can with surgery. Perfection of operative technic, shockless local anesthesia, adequate after-care are pretty generally available everywhere; yet results are not always brilliant, no matter how radical the operator may have been.

We are reminded by Stockard, Draper, Brugsch and Lewy, and other students of the human constitution that the study of human biology must take the place of the one-sided view of disease based solely on the discoveries of Pasteur and Koch.

May it not be possible that in stripping out the edematous mucosa of an antrum, substituting therefor a stratum of vascular granulation tissue, we may be performing a defensive service to the organism similar to the so-called revulsive inflammation, with "laudable" pus, which our ancients were accustomed to set up by the insertion of setons and issues? In such a layer of new tissue, mesenchymal activity, leukocytic growth and antibody formation are at a high pitch. We should remember also that sterile inflammation—artificial peritonitis set up by chemical or protein irritants—will so immunize animals that they will not succumb to subsequent injection of a hundred times the lethal quantity of hemolytic streptococci. May we not then ascribe some of the benefits of our radical operations to the defense reaction after trauma, rather than alone to the removal of purulent foci?

From another angle, if it should prove possible to create artificial febrile conditions of limited scope by diathermy, local stimulation of defense within the sinuses may be thus attempted. Certainly this has lately appealed to neurologists, some of whom have given up the treatment of paretics by malarial fever, in order to bring about similar curative effects with less danger through the artificial and controllable fever induced by electric currents.

Beneficial effects from ultraviolet radiation, from quartz lamps, arc lamps or the new General Electric S-1 mercury lamp may not improbably come from stimulation to the reticuloendothelial mechanism contained in the capillary network of the skin, by these substitutes for sunlight. Relative freedom of the naked races of mankind from sinus disease under proper diet conditions tends to affirm this theory.

We are not now discussing infective factors or imperative operative indications; nevertheless, we sometimes need to remind our friends, the internists, of maxims long ago enunciated by Skillern, Hajek and others: That the infected sinus tends to get well. We have all seen clinically positive local signs, transilluminations, X-rays, lipiodol plates: full of pus, edematous, demanding operation by all ordinary criteria. But some intercurrent ailment defers the procedure, and three or four months later the same sinus may be clear. Vaccines, a helpful crutch in overoperated or inoperable cases, do not solve this problem. Why should we be driven against our better judgment, at the behest of consultants, into radical procedures? Some of us recall the results of the radicalism of twenty-five or thirty years ago, which was not so brilliant after the lapse of five or ten years.

Hematologic study of sinus membranes after vital staining appeals to us as a fruitful field for research into the reticuloendothelial defense, and we propose to undertake such studies. Our attention is directed also to the question of increased phagocytic reaction of sinus membranes following injection of sterile oils, vital dyes and other contact methods of stimulating local immune reactions, comparable to similar studies which have been made on the pleura and peritoneum. Determination of the shifting of leukocytic elements of the blood "toward the left" should be made, to estimate the continuance of apparently healed infections or the danger of relapse. Absence of such shifting will outline the relative harmlessness of severe conditions which have been reactivated.

In all these complicated matters we must enlist the guidance of immunologists, hematologists, physiologists and dietitians, if we are not to deserve the caustic verdict of George Draper: "It is well now and then to take stock of our accomplishments

lest we become either complacent or arrogantly satisfied with what today, as well as at every past moment in our professional history, has been called modern medicine."

BIBLIOGRAPHY.

- Barlow, R. A.: Otologic and Other Manifestations of a Diet Deficient in Vitamins. *Arch. Otolar.*, 8:629, 1928.
- Baum, H. L.: Some Practical Considerations in the Problem of Sinus Infection and Drainage. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:152, 1929.
- Brugsch and Lewy: *Die Biologie der Person*. Urban & Schwarzenberg, Berlin, 1926.
- Cannon, Baer, Sullivan and Webster: The Influence of Blockade of the Reticuloendothelial System on the Formation of Antibodies. *J. Immunol.*, 17:329, 1929.
- Carscadden, W. G.: Early Inflammatory Reactions in Tissues Following Simple Injury. *Arch. Path. and Lab. Med.*, 4:329, 1927.
- Dean, L. W.: The Relation of Deficiency Diet to Diseases of the Sinuses. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:607, 1929.
- Draper, Geo.: *Human Constitution*. Saunders, Philadelphia, 1924.
- Disease: A Pysomatic Reaction, *J. A. M. A.*, 90:1281, 1928.
- Dutcher, R. A.: Vitamins in Human and Animal Nutrition. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:593, 1929.
- Emerson, F. P.: Degenerative Changes in the Lining Membrane of the Maxillary Sinus and Their Relation to Systemic Infection. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:113, 1928.
- Fenton, R. A.: Otorhinologic Pathology of Swimming. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:275, 1928.
- Gay, Clark and Linton: A Histologic Basis for Local Resistance and Immunity to *Streptococcus*. *Arch. Path. and Lab. Med.*, 1:857, 1926.
- Gay, F. P.: The Fundamental Factors of Immunity. *Medicine*, 8:211, 1929.
- Goldsmith, P. G.: The Non-Surgical Treatment of Nose and Throat Diseases. *Trans. Am. L., R. and O. Soc.*, p. 444, 1925.
- Hajek, M.: *Nebenhöhlen der Nase*. Deuticke, Leipzig, 5th ed., 1926.
- Laryngo-Rhinology and General Medicine. *Laryng.*, 39:75, 1929.
- Hill, F. T.: Hazards of Nasal Surgery in the Senile. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 36:503, 1927.
- Hudelo and Caillau: Le tissu réticulo-endothélial. *Ann. Derm. et Syph.*, 9:19, 1928.
- Ingersoll, J. M., disc. on Briggs: Evolution of the Face. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:1122, 1928.
- Jaffé, R. H.: The Reticulo-endothelial System (bibl.). *Arch. Path. and Lab. Med.*, 4:44, 1927.

Knowlton and McGregor: Regeneration of Mucous Membrane of the Maxillary Sinus. *Arch. Otolar.*, 8:647, 1928.

Kolmer, J. A.: Infection, Immunity and Biologic Therapy. Saunders, Philadelphia, 3rd ed., 1924.

King and Cocke: Therapeutic Fever by Diathermy in Paresis. *So. Med. J.*, 23:222, 1930.

Kuntz, A.: The Autonomic Nervous System. Lea-Febiger, Philadelphia, 1929.

Lewis, E. P.: Nasal Sinus Cases Treated Conservatively. *Laryng.*, 40:178, 1930.

Maximow, A. A.: Morphology of the Mesenchymal Reactions. *Arch. Path. and Lab. Med.*, 4:557, 1927.

McMahon, B. J.: Pathology of Spheno-Ethmoidal Sinusitis. *Arch. Otolar.*, 4:310, 1926.

Michele and Globus: The So-called Small Round Cell Infiltration. *Arch. Path. and Lab. Med.*, 4:693, 1927.

Mithoefer, W.: Latent Disease of the Maxillary Sinus. *Laryng.*, 39:29, 1929.

Mosher, H. P.: Unsolved Problems of Otolaryngology. *Trans. Am. O. R. and L. Soc.*, p. 354, 1924.

Mullin and Ball: Pathologic Tissues from Nasal Accessory Sinuses. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:182, 1928.

Opie, E. L.: Inflammation and Immunity. *J. Immunol.*, 17:329, 1929.

Pottenger, F. M.: The Potential Asthmatic. *J. Lab. and Clin. Med.*, 13:913, 1928.

Proetz, A. W.: The Sinus in Perspective. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:682, 1929.

Sacks, B.: The Reticulo-Endothelial System. *Physiol. Rev.*, 6:504, 1926.

Schilling, V.: The Blood Picture. Mosby, St. Louis, 1929.

Shurly, B. R.: Vitamins and Dietetics in Relation to Otolaryngology. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:251, 1928.

Deficiency Diet in Relation to the Skeleton. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:612, 1929.

Skillern, R. H.: The Accessory Sinuses. Blakiston, Philadelphia, 5th ed., 1925.

Sprunt, T. P.: Endocrine Features of Interest to the Otolaryngologist. *Arch. Otolar.*, 11:63, 1930.

Stark, W. B.: Effects of Aqueous Irrigations on the Upper Respiratory Tract of the Rabbit. *Arch. Otolar.*, 8:47, 1928.

Stockard, C. R.: Constitution and Type in Relation to Disease. *De Lamar Lectures*, J. H. Univ. Williams and Wilkins, Baltimore, p. 154, 1925-26.

The Constitution. Lane Lectures, Stanford Univ., to appear, 1930.

XXXV.

HYPERPYREXIA (FATAL) FOLLOWING
TONSILLECTOMY.

By

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Hyperpyrexia, fatal or not, is a most unusual complication following the removal of tonsils, and only a few cases are reported in the literature. It is, of course, quite possible and even probable that its occurrence is more frequent than is reported and that such tragic and untoward accidents following a smooth and technically correct operation are not reported.

A. D., male, two and a half years of age, gave a history of repeated attacks of tonsillitis. Tonsils and adenoids were removed under ether anesthesia. Operation uneventful. The child was returned to bed at 10:05 a. m., and was awake ten minutes later, crying. Pulse was of good quality. Vomited two ounces of dark blood and the temperature was 99. At 3:30 the temperature was 104. Prior to this the child had been dozing at intervals. The pulse was rapid and weak, and 300 cc. of dark blood was vomited at this time. Thinking that the child was bleeding, it was removed to the operating room by a resident physician and anesthetized with ether. The nurse's notes state that it was in poor condition; the pulse was 160 and almost imperceptible. After being put to sleep no bleeding area was found in the tonsillar fossæ. At 8:30 in the evening the temperature was 105.2, and at 2 a. m. the temperature had risen to 107.4. The pulse was still perceptible; pallor was extreme and death took place at 3:25 a. m. The urine was negative as was also the spinal fluid. No autopsy was obtained.

One other case of hyperthermia may be reported which, strictly speaking, was not postoperative inasmuch as death occurred eleven days after operation. E. J., four years of age,

whose tonsils had been removed eleven days before under ether anesthesia and who was undergoing an apparently uneventful convalescence, awoke at 2 a. m., complaining of a severe pain in the abdomen and shortly thereafter went into a convulsive state. Within four hours after the onset of the abdominal complaint the temperature was 109, the patient was unconscious and extremely pale and cyanotic. A spinal puncture demonstrated clear fluid containing five cells, all lymphocytes. The child died at 2 p. m. Temperature 105.4. At autopsy the thymus revealed marked hyperplasia of the lymphoid tissue with a moderate amount of fat and some thickening of the trabeculae. Examination of the brain revealed no abnormalities in several sections of tissue through the base, cerebrum and cerebellum. The meninges were not thickened, there were no hemorrhages in them nor any exudate. Whether this instance of fatal hyperpyrexia in a child was related to the operation under general anesthesia some eleven days before it is impossible to say; the facts are given because death occurred after tonsillectomy.

Wishart,¹ in 1909, reported the case of a girl fourteen years of age, presenting no symptoms other than those of enlarged tonsils and adenoids. The tonsils were slightly inflamed and the follicles were filled with cheesy debris. Operation was under chloroform anesthesia. There was only moderate bleeding. Following the operation the temperature rose rapidly, the pulse rate was 110, the temperature rose about one degree per hour until it reached 107. The patient vomited about a pint of bloody fluid and died about sixteen hours after the operation. Cyanosis and prostration were extreme. An autopsy gave no indication as to the cause of death. The author considered death due to violent toxemia but was puzzled as to its source.

Richardson² reported a case in 1912. The patient was a child four years old with enlarged tonsils and adenoids. When brought to the operating room Richardson was told that the temperature was 99.4, and he refused to operate. The patient was visited later in her room, and at the solicitation of the parents, who were very anxious to have the operation done, tonsillectomy was carried out. There was very little bleeding and the child was returned to her room in good condition. A few hours later it was reported that the temperature was 102,

and from that time on it rose steadily until it registered 107.4. The child died an hour after this. None of the treatment applied influenced the upward tendency of the temperature. The kind of anesthetic used was not stated. At the autopsy no blood was found in the stomach or intestines, and all of the organs of the body presented a normal, gross and microscopic, appearance. No cause of death could be ascertained.

Dabney³ reports a case of hyperpyrexia following tonsillectomy which, however, did not end fatally.

The patient was an adult of good habits. The tonsils were removed under the handicap of a badly given anesthetic, there never being sufficient relaxation to dispense with the help of an orderly. There was furthermore much difficulty due to bleeding. The temperature rose from normal at the beginning of the operation to 102 in two hours, and four hours later it was 105. There was mild delirium for twenty-four hours with a gradual return to normal.

It is questionable if the hyperpyrexia in these cases was due to the particular operation (tonsillectomy), as this complication is occasionally observed after various other operations, especially in children. The literature shows a number of such cases.

Makai⁴ reported two fatal cases. The first was in a child ten months of age, operated on under ether anesthesia for hernia. Following the operation the temperature rose to 41.3 C. (about 106 F.), and was accompanied by the exceptional pallor. Death occurred sixteen hours after the operation, which had been uneventful. An autopsy revealed a high grade hyperemia and edema of the brain and spinal cord. There was no evidence of sepsis.

The second case was in a child two and one-half years of age, operated on under ether anesthesia for cleft palate. The child was pale during the operation, following which the temperature rose to 40.7 C. (about 105 F.), with convulsions, and death occurring sixty hours after the operation. An autopsy revealed hyperemia of the brain and meninges and no sign of sepsis.

Armingeat⁵ reports a case of a child, nine months of age, operated on under ether anesthesia for a small tuberculous lesion in a shoulder region. The temperature at the beginning

of the operation was 37.2 C. (99 F.). The child awoke pale: the pulse was rapid. The temperature rose gradually and seventeen hours after the operation it was 40.4 C. (105 F.). Although death seemed imminent, the child recovered.

Gierthmühlen⁶ reports a fatal case in a child one year of age, operated on under chloroform anesthesia for adenocarcinoma of the kidney. The temperature rose to 39.4 and the child died nine hours after the operation.

Canuyt and Terracol⁷ report a fatal case. A child, eight months of age, was operated on under chloroform anesthesia for a postauricular subperiosteal abscess following otitis media. In the afternoon following the operation the child became extremely pale. The temperature was 40 C. (104 F.) and the pulse 140. Death occurred a few hours later, the temperature having risen to 42 C. (107 F.). An autopsy revealed no adequate cause for death. The authors say that several such deaths occurred in their hospital in the experience of several different operators and in precisely the same way, without autopsies revealing an adequate cause for death. Furthermore, they claim that this syndrome of extreme pallor and hyperpyrexia followed quickly by death is not unknown to most surgeons in children's clinics. They find it impossible to foresee when it may occur.

Besides his fatal cases, Makai reported another in which the temperature rose to 40.1 C. (104 F.), within eight hours after an operation for the cure of harelip. In this instance the child recovered. Gierthmühlen, too, aside from his fatal case, observed six others with recovery in which hyperpyrexia followed operations in children.

Ingelrans and Minne,⁸ in a recent article, report two fatal cases. The first concerns a male child, one year of age, operated on for a left sided strangulated inguinal hernia. The operation was carried out under general anesthesia and strict preservation of asepsis. The temperature at the time of operation was 37.5 C. (99.8 F.). That evening, seventeen hours later, the temperature rose to 39.5 C. (103 F.). It continued to rise steadily the following day until it reached 41.5 C. The pallor was intense. Death occurred on the third day after operation. An autopsy revealed marked congestion of the brain with hemorrhage in the cortex and in the lenticulostriate region. The

choroid plexus was markedly injected and there was a slight degree of distension of the lateral ventricles.

The second case concerns a child, two and one-half months old, operated on for a strangulated hernia. Death occurred six hours following the operation, with the temperature at that time 40.6 C. (105 F.). Extreme pallor supervened three hours after the operation. There were no other noteworthy symptoms. At autopsy there was found only intense congestion of the brain accompanied by a mild external and internal hydrocephalus.

It is clear from a review of the literature that there is a variation of opinion as to the cause of postoperative hyperpyrexia.

It has been blamed on the anesthetic. It is well known that severe reactions occasionally follow anesthesia. Tawse⁹ reported a case of tonsillectomy in a boy, two years of age, under ethyl chloride anesthesia. There was only slight hemorrhage and the child slept well the following night. Early the next morning death occurred, preceded by sudden marked pallor and collapse. There is no mention of hyperpyrexia. An autopsy showed a hemorrhage into the pons the size of a peanut. Everything else apparently was normal. McCardie and Featherstone¹⁰ also report two cases of sudden death from hemorrhage into the pons following general anesthesia. They do not mention the presence of hyperpyrexia. Atkeison¹¹ reported a case in which, following nitrous oxide and oxygen anesthesia, there was prolonged unconsciousness (thirteen days), with a temperature in the neighborhood of 103.4 F. At the autopsy no examination was made of the cranial contents.

Fetteroli¹² says that following tonsillectomy the temperature ranges from 99 to 101, rarely going higher, unless there is a definite complication such as suppurative otitis media.

Drevermann¹³ and also Gierthmühlen think that latent spasmodophilia may be responsible for hyperpyrexia following a faultlessly executed operation. They say that infection may be excluded, for the fever is too sudden in its onset to be accounted for by this cause. Nor do these patients exhibit the usual picture of high fever. They are very pale and the facies resembles the Hippocratic type. Makai also agrees that postoperative hyperpyrexia is independent of narcosis; that it can

occur whether narcosis is used or not; that the duration of the operation is not a factor; and he seems to feel that spasmodophilia or status lymphaticus plays a rôle.

Ombredanne,¹⁴ in his "Chirurgie Infantile," 1925, states that sudden death of operated on infants has occurred in his experience, accompanied by pallor and hyperpyrexia; that this often happens within twenty-four hours following operations, and that it occurs mostly in infants and rarely after the second year.

The hyperpyrexia accompanied by pallor may reach 42 C. (107.5 F.) within five or six hours after operation. Should this happen so soon, the prognosis is usually fatal. Death is exceptional if the hyperpyrexia lasts longer than twenty-four hours. This syndrome, according to Ombredanne, has been observed not only following major but also following trivial operations, such as those for the cure of phimosis. But it would appear that operations on the face and for the relief of harelip are more likely to have this complication, especially in children with some degree of hydrocephalus.

This accident when it has occurred has been considered by some as due to a fulminating bronchopneumonia or as an acute pulmonary edema consecutive to anesthesia. It has also been thought by some that blood swallowed during an operation in the pharynx has been a factor, this blood acting as a foreign toxic protein. But this explanation will not apply when this accident has happened following operations away from the oral cavity. Ombredanne thinks that none of the hypotheses advanced up to the present time are adequate. He seems to favor the idea that the anesthesia or operative shock severely injure important organs, such as the liver, the adrenal glands and the thymus. The result would be a sensitization of the organism somewhat analogous to anaphylactic shock.

Elizalde¹⁵ thinks that the cause of postoperative hyperpyrexia in children is due to blood loss, dehydration after vomiting and lack of water intake.

Armington¹⁶ has made a critical study of the various hypotheses put forward, taking into account the physiologic aspects, the alkali reserve, acidosis, dehydration, etc. He has found that there is a rapid and marked fall in the arterial tension at

the moment of maximum temperature. He reasons that the operative act normally provokes some fever in the infant, which has a poor heat regulating apparatus. It does not perspire, has no chills and its only defense is polypnea, by which there is a great water loss and consequent hyperthermia. Armingeat likewise thinks that the poor heat regulation mechanism is concerned in the dehydration, and he assigns the pallor to bulbar excitation, which produces a peripheral vasoconstriction.

Ingelrans and Minne,⁸ in their recent paper discuss the various etiologic theories and point to the extreme congestion of the brain in their own two cases, accompanied by external and internal hydrocephalus. Autopsy in their experience furthermore failed to reveal any other outstanding findings. They advance a new theory, namely, the unstable nervous system in children, lacking the normal reflexes of defense, may be aroused by even insignificant operative traumatism to fulminating defense responses.

Considering the unsettled notions as to the etiology of these symptoms of pallor, hyperpyrexia and occasionally death following anesthesia and operations, there can be no logical prophylactic measures. It would seem wise not to operate on any child with a preoperative temperature above 99.2; to avoid general anesthesia if possible, and to reduce operative manipulations to a minimum.

BIBLIOGRAPHY.

1. Wishart, J. G.: Hyperpyrexia and Death Following Tonsillectomy. *Canada Lancet*, 1909-10, XLIII, 111.
2. Richardson, C. W.: Complications of the Operation for Removal of Tonsils. *Trans. Amer. Laryngol. Assn.*, 1912, XXXIV, 170.
3. Dabney, V.: *Transac. Amer. Laryngol. Assn.*, 1913, XXXV, 282.
4. Makai, E.: Eine rasch verlaufende, zumeist tödliche Komplikation nach Säuglings-Operationen. *Münch. med. Wechschr.*, 1928, LXXV, 511.
5. Armingeat: Sur un cas de pâleur avec hyperthermia chez un nourrisson opéré. *Bull. Soc. de Pédiat., Paris*, Oct., 1928, XXVI, 422.
6. Gierthmühlen, F.: Hyperpyrexie nach Säuglings-Operationen. *Münch. med. Wechschr.*, 1928, LXXV, 1604.
7. Canuyt, G., and Terracol, J.: La mort rapide du nourrisson et les interventions sur l'antre mastoïdien. *Rev. de laryngol. etc., Paris*, 1925, XLVI, 437.
8. Ingelrans, P., and Minne, J.: *Presse méd. Paris*, II, Sept., 1929, p. 1184.

9. Tawse, H. B.: An Unusual Case of Death after the Removal of Tonsils. *Jour. Laryngol.*, 1926, XLI, 97.
10. McCardie, W. J., and Featherstone, H. W.: *Lancet*, London, 1926, I, 810.
11. Atkeison, J. E. H.: *American J. Surg.*, Jan., 1923, XXXVII, (Anesth. Supp.) 17-18.
12. Fetterolf, G.: *Amer. Jour. Med. Sc.*, CLXIV, 884.
13. Drevermann: *Ergebn. d. Chir. und der Orthopädie*, 1925, Bd. XVIII.
14. Ombredanne, L.: *Chirurgie Infantile*. Paris, 2. Edit., 1925, p. 7.
15. Elizalda, P. de: *Semana méd.* Buenos Aires, 1919, XXVI, 404.
16. Armingeat: Thesis on Ombredanne's presentation of this subject at the Montreal Congress, 1922.

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XXXVI.

ALLERGY AND ITS RELATION TO THE INFLAM-
MATORY DISEASES OF THE PARANASAL
SINUSES.*

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An understanding of the relationship of allergy to the inflammatory diseases of the paranasal sinuses entails the clinical and histopathologic study of vasomotor or hyperesthetic rhinitis, hay fever, bronchial asthma, catarrhal rhinitis and sinusitis, nasal and sinus polypi and edema, and hyperplastic ethmoiditis.

In 1910, Auer and Lewis¹ found that the lungs of guinea pigs that died in anaphylactic shock became distended because of bronchospasm. On the basis of this observation, Meltzer² called attention to the similarity of the symptoms and lesions to those of bronchial asthma. In 1906, Wolff-Eisner³ had already suggested that hay fever was anaphylactic in nature. The use of the skin test in the establishment of pollens as the cause of hay fever was first demonstrated by Blackley⁴ in 1865. The application of the skin test to foreign protein substances was instituted about twenty years ago by Noon,⁵ Cooke,^{6 7} Schloss^{8 9} and Walker.^{10 11} Bronchial asthma came to be considered one of the important clinical expressions of anaphylaxis in man. With the establishment of allergy as the etiologic factor in bronchial asthma and hay fever, it was soon discovered that nonseasonal hay fever or vasomotor rhinitis was also allergic in nature.

About ten years ago I¹² made a study of vasomotor rhinitis with the idea chiefly of determining the etiologic factors. It

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was found that in a group of 100 cases more than 50 per cent were allergic as determined by skin tests. The apparently non-allergic cases were considered as reflex types. There was no difference, however, in the two types of cases from the standpoint of symptoms and nasal findings. A continuation of these studies with most careful consideration of the allergic phases resulted in a much better understanding of the importance of allergy as an etiologic factor. In a recent report¹³ on "Clinical and Histopathologic Studies of the Nose and Sinuses in Allergy," based on a study of more than 1,000 cases, it was established that the above enumerated conditions should be considered as primarily of allergic origin.

If we consider the progress of allergic disease of the nose and sinuses from its onset to the advanced stages of pathologic change, we shall note that the various terms enumerated above merely represent different stages of the same disease. An analysis of the terminology shows that there has been a tendency to designate the early type of case as vasomotor or hyperesthetic rhinitis, the more advanced case perhaps as catarrhal rhinitis, catarrhal sinusitis or hyperplastic ethmoiditis, and the advanced case as one of nasal and sinus polypi or as nonsuppurative or hyperplastic sinusitis.

The clinical and histopathologic picture is not always classical, for consideration must be given to the fact that the patient may be temporarily free of symptoms or in a negative phase, or that he has been entirely free of allergic manifestations for a variable period of time. It must also be remembered that the occurrence of acute infection or the establishment of chronic secondary infection frequently complicates the diagnostic problem.

In order to establish the diagnosis of allergic disease of the nose and sinuses a routine method of investigation should be adopted. It is suggested, therefore, that the following factors be considered: (1) Nasal symptoms, (2) rhinoscopic examination, (3) examination of nasal secretions, (4) roentgenographic study of sinuses, and (5) histopathologic examination of nasal and sinus tissues. A study of the allergic state of the patient should be made with particular reference to: (1) The occurrence of other manifestations of allergy, (2) a positive

family history of allergy, (3) the presence of positive skin tests, and (4) blood eosinophilia.

Briefly the nasal manifestations of allergy are characterized symptomatically by sneezing, nasal obstruction and nasal discharge. In hay fever these symptoms are frequently well marked and easily recognized. In the other or nonseasonal types, the symptoms are generally intermittent, vary considerably in degree and are apt to be confused with frequent colds.

The nasal discharge in allergy varies from thin serous liquid to thick, tenacious mucus, and the microscopic examination usually shows the presence of eosinophiles.

The examination of the nose in allergy reveals pathologic changes which are usually strikingly characteristic. The membrane appears discolored grayish-pink or bluish-gray, with swelling or edema. Edema is particularly apparent in the middle meatus or anterior ethmoid regions. In cases with more advanced pathology, hyperplasia and polypoid formations may be observed. The nose may be filled with polypi which are attached usually in the anterior and posterior ethmoid regions. These formations are the result of sagging or prolapse of the edematous mucosa. The examination of the sinuses, particularly the ethmoid cells and the maxillary antrums, may show edema, hyperplasia and polypi as found in the nose.

In the histologic examination of the nasal tissues the average mucosa contains a certain relative amount of connective tissue, lymphocytes, other lymphoid cells, eosinophiles, blood vessels, cavernous vessels, glands, lymphatic and distended interstitial spaces. Finck¹¹ has shown that in definitely abnormal states the nasal mucosa presents relative increase or decrease in these tissue elements under the epithelium—that is, in the tunica propria or stroma. The epithelium may show thickening, hyperplasia or loss of substance.

The histopathologic examination of the nasal and sinus tissues in allergy shows pathologic changes similar to those found in the bronchial tissues in bronchial asthma. The surface epithelium is thickened and consists of many layers of stratified columnar cells. Certain areas may be observed where thickening is not sufficiently marked to cause loss of cilia. Areas of desquamation of epithelium appear scattered here and there.

The subepithelial tissues are loose and edematous and infiltrated with eosinophiles which are usually found in the tissues in allergy. Marked accumulations frequently occur just under the epithelium. Other cellular elements consist mostly of plasma cells and lymphocytes. Large tissue spaces contain serum which gives the edematous appearance. The glandular structures usually appear dilated and filled with secretion. Large cystic areas lined with epithelium represent dilated glands or gland ducts. In advanced stages atrophy and disappearance of glands may occur. Cellular accumulations appear around blood vessels and glands. On the whole, the cellular reaction is much less but the edema is more marked than that seen in true inflammatory processes. The typical pathologic picture of allergy is confined chiefly to the subepithelial layer of the mucosa and usually extends partly into the glandular layer. Edema and eosinophilic infiltration rarely extend deeper than the glandular layer. Differential cell counts in edematous areas show, on the average: eosinophiles 30 to 40 per cent, lymphocytes 35 to 45 per cent, and plasma cells 20 to 30 per cent. The number of eosinophiles in the blood, the tissues and consequently also in the nasal secretions may show variations in different cases. Even in the same specimen there may be considerable variation in the distribution of cellular elements. Eosinophiles may be scarce in some areas and very densely collected in other areas. Similar variations occur in the distribution of lymphocytes and plasma cells. Edema may be more pronounced in some areas than others. Variations in the cellular elements exclusive of the eosinophiles may be dependent upon the age of the patient, the occurrence of frequent attacks of acute infection or upon the chronicity of the allergic process. Bone changes are confined practically to the ethmoid cells, where there is a tendency to rarefaction. Bone changes here are frequently absent, and when present are relatively insignificant unless well pronounced.

Roentgenographic examination of the paranasal sinuses in allergy frequently shows varying degrees of opacity, depending upon the degree of pathologic change and the presence or absence of retained secretions. Edema and polypi frequently may be shown by the use of lipiodol. Transitory edema may occur, giving rise to positive findings at one time and

negative findings at another time. Dean, Proetz and also Johnston¹⁵ have also proved recently that this variation may occur. Transitory edema in the antrum usually shows an irregular surface outline in the lipiodol plate, while the more established chronic edema is apt to show a rather smooth surface outline.

The pathologic condition of the nose and sinuses thus resulting from allergic disease prepares the soil for infection. The edematous mucosa, with the loss of cilia which under normal conditions keep the sinuses free of secretions and bacteria, causes the retention of these products. Stagnation of secretions readily occurs and with added attacks of acute infection a definite suppurative process tends to become active and eventually may result in chronic infection.

The histopathologic examination of the nasal and sinus tissues in allergy with secondary infection shows a marked increase in the degree of cellular reaction. In the acute stage the polymorphonuclear leucocyte is the most predominant cell with a relative increase in the number of lymphocytes or round cells. In the chronic process the round cell and plasma cells are most predominant. The proliferation of new connective tissue is also an outstanding feature. Cellular accumulations in acute and chronic processes may be of sufficient degree to cause localized abscesses in the tissues. It is usually possible, however, to find areas in the tissues where the picture of allergy remains with little alteration. The presence of mucopurulent secretion in these cases tends to obscure the allergic background, but a careful investigation of the clinical course and the histopathologic examination of the tissues should enable one to recognize the allergic relationship.

The classification of the inflammatory diseases of the paranasal sinuses, as accepted by otolaryngologists today, is as follows:

1. Suppurative paranasal sinusitis.
 - a. Acute empyema.
 - b. Chronic empyema.
 1. Edematous type.
 2. Granular type.
 3. Fibrous type.

2. Nonsuppurative or hyperplastic paranasal sinuses, including acute and chronic catarrhal sinusitis.
3. Combined type, suppurative-hyperplastic sinusitis.

According to this classification, no distinction is made between allergic and nonallergic sinus disease. I believe that allergic disease of the sinuses should be accepted as a definite clinical entity and should not be considered in the same light as primary suppurative disease. Although the occurrence of secondary infection in allergic cases places them in a class with the suppurative diseases, even in this instance the primary occurrence of the allergic process so greatly influences the prognosis and treatment to such an extent that the two groups of cases should be considered separately if satisfactory results are to be obtained. Dean¹⁰ has emphasized the importance of always considering the possibility of allergy in the diagnosis of sinus disease in children.

Many of the chronic empyemas undoubtedly follow in the wake of an allergic process in which an edema of the mucosa has occurred. In the nonsuppurative, hyperplastic or catarrhal group of paranasal sinus diseases, clinical and histopathologic studies have proved that in the great majority of cases the process is primarily an allergic disease. Therefore all hyperplastic, nonsuppurative sinus disease should be considered as of allergic origin until proved otherwise. A detailed investigation of the symptoms, of the nasal secretions for eosinophilia, of the histopathology of the tissues and of the allergic state of the patient will establish the diagnosis.

In the combined group, the suppurative-hyperplastic type, the allergic must be distinguished from the nonallergic. Suppurative-hyperplastic sinusitis may be either allergy with secondary infection or true primary suppurative sinusitis with secondary hyperplasia. Nasal polypi occur in both types. They occur more frequently and abundantly in allergy than in primary infection. We must recognize them, therefore, as occurring, primarily, in one instance as the result of allergy and in the other instance as the result of infection. Certain allowance should be made, however, for a small percentage of polyp cases in which apparently neither allergy nor infection plays a part. These are the cases in which the etiologic factor is

apparently a chronic nonspecific irritation such as dust, chemicals and other like substances. It is also possible that individual tendencies or constitutional factors may also play a part.

In primary sinus infection or suppuration there is a striking absence of vasomotor symptoms, and upon examination of the nose the mucous membrane may appear inflamed and sometimes edematous. Polypi if present appear injected and more firm or compact than seen in allergy. When the nose is markedly obstructed with polypi in allergy the vasomotor nasal reactions may be absent or infrequent on account of the lack of nasal respiration. The history will usually disclose, however, the previous existence of these symptoms and other manifestations of allergy. In cases of doubtful diagnosis one should not hesitate to remove tissue for microscopic diagnosis.

The histopathologic examination of the tissues in primary infection of the sinuses shows, in addition to hyperplasia of the surface epithelium, a marked cellular infiltration of the subepithelial tissues consisting of polymorphonuclear leucocytes, round cells and plasma cells. In acute cases or in acute exacerbations in chronic cases the polynuclears are more abundant, while in the usual chronic state the round cell is predominant and connective tissue proliferation is very active.

On the basis of clinical and histopathologic observations it is apparent that the allergic sinus cases, with or without secondary infection, should be distinguished from the true infections or primary suppurative sinus cases. I believe that many of the disappointments in the treatment of sinus disease have been the result of the failure to make this distinction.

Having considered the relationship of allergy to the inflammatory diseases of the paranasal sinuses it is now interesting to review the literature and note the observations and opinions of various observers on the subject of nasal polypi.

In recent publications Hirsch^{17, 18} reviewed the literature on the subject of nasal polypi. He began with a description of the work of Billroth, in 1855, and discussed in chronologic order the studies and opinions of other outstanding authorities up to the present time. The studies of Zuckerkandl, Heymann, Hopmann, Hajek, Cholewa, Cordes, Woakes, Uffenorde and others were reviewed in detail. Some observers believed

that the edematous type of polyp originates primarily in the mucous membrane of the ethmoid surface, lining of the ethmoid cells and surface of the middle turbinate. Others believed that these polypi originate from disease of the ethmoid bone. Another group maintained that polypi originate from infection chiefly in the ethmoid cells and maxillary antrums. From Hirsch's own observations he concludes that they originate as a result of a chronic catarrhal inflammation of the sinuses, principally the ethmoid cells and maxillary antrums.

The histologic character of mucous polypi was first established by Billroth,¹⁹ and his findings were later confirmed by other observers. Hopmann^{20, 21, 22} established the fact that the polyp consists of a network of areolar connective tissue but stated that he never found the gland formations described by Billroth.

Zuckerkandl²³ made the important distinction of considering genuine nasal polypi as a type separate and distinct from other soft swellings in the nose, and believed that they are inflammatory products of the mucous membrane. He pointed out that the relation of stroma and exudate in nasal polypi is just the same as in that form of disease of the mucous membrane of the maxillary sinus which he designated as catarrhal. Zuckerkandl also emphasized that polypi occur principally in the respiratory zone of the nasal cavity, namely, (1) on the edges of the hiatus semilunaris, (2) in the infundibulum, (3) at the ethmoid ostia, (4) at the ostium frontale, (5) at the ostium maxillare, (6) on the edge of the middle turbinate, (7) on the edges of the accessory furrows on the medial side of the middle turbinate, (8) on the ethmoid bulla, and (9) in the ethmoid cells. He attributed their origin to a superficial inflammation of the ethmoid region and called the process a rhinitis. Papillomata and polypoid hypertrophies, such as those which form especially on the posterior tips of the turbinates, were not considered with the genuine nasal polyps.

Hopmann²² divided soft growths occurring on the turbinates and the lateral wall into three groups: (1) edematous fibroma or mucous polyp, (2) polypoid hyperplasia or glandular polyp, and (3) papilloma or mulberry polyp (papillary hypertrophy).

With Zuckerkandl's discovery that polypi are of an inflammatory nature, the question of the origin of polypi came into

the foreground. At the beginning of the twentieth century, general constitutional predisposition and heredity were generally accepted as the causes of the formation of nasal polypi. Uffenorde^{24 25 26} has always maintained the special significance of constitutional predisposition. In addition to these general causes, the old authors frequently found pus in the nose when filled with polypi, and they assumed that polypi were primary and the suppuration the consequence of irritation by the polypi. Up until the time of Zuckerkandl it was generally believed that polypi were the consequence of catarrh of the nose. In 1885 Woakes²⁷ provoked considerable discussion by making the statement that nasal polypi were the result of necrosing ethmoiditis.

In 1896 Hajek²⁸ described the inflammatory affections of the mucous membrane of the ethmoid as occurring in three forms: (1) superficial inflammations, (2) deep inflammation which involves the periosteum and medullary spaces of the bone, and (3) apposition and rarefaction of the bone. Hajek maintained that the inflammation begins on the surface and extends more deeply to the bone. The studies of Hajek were confirmed and recognized by all later investigators. In 1900 Cholewa²⁹ regarded the formation of nasal polypi as the result of subacute osteomyelitis. In 1901 Cordes³⁰ confirmed the findings of Hajek but did not find the rarefying osteitis described by him.

In 1907 Uffenorde²⁴ also agreed with Hajek and called the changes of the ethmoid mucous membrane which lead to polyp formation a hyperplastic ethmoiditis. About the same time E. S. Yonge³¹ expressed the opinion that polypi are the result of local circulatory disturbances, and Watson-Williams³² attributed the cause to an infectious lymphangitis.

About this time (1907) two chief theories as to the origin of polyps were considered. That is, they were caused, according to one theory, by an inflammation of the mucous membrane of the middle meatus, and according to the other theory they arose from a bone affection. In 1893 Grünwald³³ advanced the idea that polypi were pathognomonic of sinus suppuration. In 1896 A. Alexander³⁴ found that some polypi cases were not associated with sinus suppuration and others were associated with sinus suppuration. Hajek³⁵ also emphasized

that polypi may or may not be associated with suppuration. He also refers to the fact that polypi have a tendency to recur in nonsuppurative cases and that frequently in suppurative cases the polypi do not have a tendency to recurrence if the suppuration is cured.

The conclusion was finally reached that there was no uniform cause for polypi and that they could either arise primarily as polypoid or benign degeneration (Zuckerkanhl, Hajek, Uffenorde) or as secondarily in consequence of the irritation of pus flowing from the sinuses (Grünwald) or by extension of the inflammatory infiltration of the sinus membrane through the sinus opening to the middle meatus (Alexander).

Recurrence of nasal polypi was explained at this time: (1) as the result of imperfect removal of polypi, (2) as the result of deep inflammation involving the bone marrow, (3) following neglected sinus empyema, (4) in rare cases from polypoid degeneration of the lining of the accessory sinuses (Hajek).

After long continued observation, leading up to only a few years ago (1923), Uffenorde³⁶ concludes that polypi originate from the nasal surface of the ethmoid and extend into the ethmoid cells and through the bony walls to the other sinuses. He also concludes that these inflammations are the cause of recurrence of polyps which continue to reform unless all diseased membrane is removed.

Hirsch,^{17 18} on the basis of his observations beginning in 1919-1920, states that recurring polypi have their origin from the first in a catarrhal inflammation of a sinus, most often the maxillary antrum. He maintains that the origin of nasal polypi may be explained on the basis of the formation of choanal polypi, which most frequently arise in the antrum. On this basis he concludes that recurring polypi originate in the beginning from the sinuses and only as a result of catarrhal inflammation of the sinuses. Hirsch's final conclusions are:

"1. Polypi are partly prolapses of the catarrhal mucous membrane of the sinuses, principally that of the antrum, and arise by incarceration of peaks of mucous membrane or by a continuation of the catarrhal inflammation of the sinus (principally the antrum) by way of the ostium of the sinus.

2. From the presence of polypi, especially such as show inclination to recurrence, and from the site of the polypi, a catarrhal inflammation of the affected cavity can be diagnosed, just as a purulent inflammation of the affected sinus from the finding and localization of the pus streak."

Hirsch divides polypi into two groups, primary polypi and secondary (recurring) polypi. Primary polypi, he states, arise on the anterior end of the middle turbinate and in accessory furrows of the middle meatus. These are the polypi which arise a primary catarrhal rhinitis. He therefore calls them primary polypi. He also asserts that these show little or no tendency to recur, while polypi which arise from the sinuses regularly recur so he calls them secondary or recurring polypi.

In a consideration of nonsuppurative sinusitis based on the principles of Hirsch, Fraser³⁷ reported 120 cases of chronic catarrhal sinusitis selected at random from 350 cases examined by the use of lipiodol. These cases showed thickening without pus or with pus only as an acute accompaniment. Diagnosis was also based upon such symptoms as sneezing with watery discharge, obstructed nasal polypi, and the sudden onset of symptoms without the usual history of coryza.

Fraser found the ethmoid more frequently involved than the maxillary antrum in bilateral cases. Hirsch, on the other hand, found the antrum most frequently involved. Fraser outlines the rules for classification of sinus involvement by means of nomenclature and the clinical findings. He also suggests a classification along the lines of therapy and divides the cases into six types. While the work of Fraser is very commendable for the excellent manner in which the cases are classified, it is unfortunate that pathologic studies were not carefully considered and correlated with the clinical, X-ray and operative findings. It is also unfortunate that allergic nasal and sinus disease was not considered as a factor in this group of cases, for it is quite evident that the outstanding occurrence of vasomotor symptoms suggests a high incidence of allergic disease.

† The belief among many of the old authors that nasal polypi occurring chiefly with a catarrhal rhinitis and sinusitis, in the absence of suppuration, were greatly dependent upon constitutional predisposition and heredity, were more correct in their

conceptions than the more recent observers. In the symptom groups which Uffenorde used as a diagnostic basis for the occurrence of hyperplastic ethmoiditis, he emphasizes such symptoms as frequent colds, continual feelings of cold, obstruction and watery discharge. The frequency with which bronchial asthma was found among the complaints led him to believe that the changes in the sinuses were important factors in causing the asthma. With our present understanding of allergy as a factor in nasal and sinus disease, we know that the constitutional and hereditary factors referred to were allergy.

The classification suggested by Hirsch does not consider allergy as an etiologic factor. We should consider the nasal polypi occurring in vasomotor rhinitis, hay fever, bronchial asthma, acute and chronic catarrhal rhinitis and sinusitis, non-suppurative hyperplastic sinusitis (ethmoiditis), and certain combined types of sinusitis as primarily of allergic origin. Every possible method of investigation of the allergic factors as already mentioned should be carried out.

In the consideration of ordinary nasal polypi (edematous and glandular), therefore, we should distinguish those of true allergic origin, those of unproved but apparently of allergic origin occurring with or without secondary infection, from those polypi which occur secondarily from a primary suppurative sinusitis, or from chronic irritation factors. In this entire consideration of nasal polypi the true choanal polypi, mulberry hypertrophies and certain papillary hypertrophies should be excluded.

Among the numerous histopathologic reports in the literature on the study of nasal polypi it is remarkable to note that very little attention and significance was attached to what we now recognize as a typical allergic reaction of the tissues. The occurrence of the eosinophilic infiltration in edematous polypi has been only casually mentioned among older authors. Although constitutional predisposition and heredity were mentioned as causes and we now interpret these causes as allergic, eosinophilic infiltration was not mentioned in this connection.

In 1895 Seifert and Kahn,³⁸ and in 1900 B. Lewy³⁹ gave an accurate description and exhibited beautiful illustrations of what we now consider a typical histopathologic picture of

allergy. They called attention to the occurrence of this histopathologic change in the nasal tissues in bronchial asthma, in acute and chronic catarrhal rhinitis and in nasal polypi of the edematous type. They also demonstrated the occurrence of Charcot-Leyden crystals in nasal polypi associated with eosinophilic infiltration. A few years ago (1921) Jacobsthal⁴⁰ demonstrated in a study of eosinophil cells that the eosinophilic granules are extruded from the cells into the tissues and that Charcot-Leyden crystals are formed from these granules.

In 1927 Finck⁴¹ called attention to the occurrence of eosinophilic infiltration of the nasal tissues in vasomotor rhinitis, bronchial asthma and in certain nasal polypi. Mullin and Ball⁴² also made this observation in cases of bronchial asthma. Israel⁴² recently mentioned the occurrence of eosinophilic infiltration of the mucosa in certain cases of maxillary sinusitis.

In Henke and Lubarsch's⁴³ "Handbook of Special Pathologic Anatomy and Histologie," reference is made to the occurrence of eosinophilic infiltration in the tissues in acute and chronic catarrhal rhinitis and in nasal polypi, but its possible association with allergy is not mentioned.

A most interesting report on allergic rhinitis (hay fever excluded) is that of Leicher.⁴⁴ This work is an important example of how our cases of nasal and sinus allergy should be investigated. He divides his cases into two groups, as follows: Group 1, Vasomotor rhinitis; Group 2, Polypoid, nonsuppurative (serous) nasal and paranasal sinus inflammation. In the vasomotor rhinitis group there were 31 cases, 28 (90 per cent) of which were definitely of allergic origin. In the nonsuppurative polypoid group there were 38 cases of which 13, or 34 per cent, were of allergic origin.

The allergic origin of these cases was based on the following symptoms:

1. Blood eosinophilia, eosinophiles in the nasal secretions and local tissue eosinophiles in the turbinates, nasal polypi and mucous membrane of the sinuses.
2. Positive skin tests.
3. Elevation of K/Ca quotient in the blood; the so-called vagotonia-adrenalin-blood-pressure reaction; alteration of calcium

excretion in the blood and urine after intravenous calcium injection.

4. Positive family history of allergy.

In group 1, 28 of the 31 cases showed a blood eosinophilia varying from 4 to 16 per cent. In two cases in which repeated examinations showed a blood eosinophilia, an aneosinophilia or negative phase was noted immediately after an attack of vasomotor rhinitis. In this group eosinophiles were frequently found in the nasal secretions.

In group 2, 13 of the 38 cases showed a blood eosinophilia, and 8 cases showed eosinophiles in the nasal secretions. Tissue eosinophilia was demonstrated in 6 cases in group 1 and in 13 cases in group 2.

The results of skin tests in group 1 showed 28 positive and 3 negative. In group 2 there were 34 positive and 4 negative reactions.

Other studies in these patients showed an elevation of the K/ca quotient in the blood serum of both groups. In the so-called adrenalin blood pressure reaction there was a primary fall of blood pressure (5—10 mm. Hg.) in the majority of cases. There was also an alteration of calcium excretion from the blood and urine after intravenous injection of calcium.

An investigation of the family history in the 69 cases, 25 were positive. Other manifestations of allergy in the same patient occurred in 13 cases.

CONCLUSIONS.

1. An understanding of the relationship of allergy to the inflammatory diseases of the paranasal sinuses entails the clinical and histopathologic study of vasomotor or hyperesthetic rhinitis, hay fever, bronchial asthma, catarrhal rhinitis and sinusitis, nasal and paranasal polypi and edema, and hyperplastic ethmoiditis.

2. The diagnosis of allergic disease of the nose and sinuses should be based upon the follow factors: 1, Nasal symptoms; 2, rhinoscopic examination; 3, examination of nasal secretions for eosinophiles; 4, roentgenographic study of the sinuses, and 5, histopathologic examination of nasal and sinus tissues. The allergic state of the patient should be studied with reference to: 1, The occurrence of other manifestations of allergy; 2, a

positive family history of allergy; 3, the presence of positive skin tests, and 4, blood eosinophilia.

3. Most nasal polypi should be considered as being either of allergic origin or as secondary to a suppurative process.

4. Nonsuppurative or hyperplastic sinus disease should be considered as primarily of allergic origin until proved otherwise.

5. Allergic sinus disease, with or without secondary infection, should be differentiated from primary sinus infection with edema, hyperplasia or polypi.

REFERENCES.

1. Auer, J., and Lewis, P. A.: The Physiology of the Immediate Reaction of Anaphylaxis in the Guinea-pig. *Jour. Exp. Med.*, 12:151, 1910.
2. Meltzer, S. J.: Bronchial Asthma as a Phenomenon of Anaphylaxis. *Jour. Am. Med. Assn.*, 55:1021, 1910.
3. Wolff-Eisner, A.: *Das Heufieber: Sein Wesen und Seine Behandlung*. Munich, 1906.
4. Blackley, C. H.: *Hayfever. Its Causes, Treatment and Effective Prevention*. London, 1880, 2nd Ed., p. 93.
5. Noon, L.: Prophylactic Inoculation against Hayfever. *Lancet*, 1:1572, 1911.
6. Cooke, R. A.: The Treatment of Hayfever by Active Immunization. *Laryngoscope*, 25:108, 1915.
7. Cooke, R. A., and Vander Veer, A. Jr.: Human Sensitization. *Jour. Immunol.*, 1:201-306, 1916.
8. Schloss, O. M.: A Case of Allergy to Common Foods. *Am. Jour. Dis. Child.*, 3:341-362, 1912.
9. Schloss, O. M.: Allergy in Infants and Children. *Am. Jour. Dis. Child.*, 19:433-454, 1920.
10. Walker, I. C.: Studies on *Staphylococcus Pyogenes Aureus*, *Albus* and *Citrens*, etc. *Jour. Med. Res.*, 35:373-390, 1916.
11. Walker, I. C.: Studies of a Diptheroid Organism Isolated from the Sputum of Patients with Bronchial Asthma. *Jour. Med. Res.*, 35:391-402, 1916.
12. Hansel, F. K.: Vasomotor Rhinitis. *Jour. Am. Med. Assn.*, 62:15-17, 1924.
13. Hansel, F. K.: Clinical and Histopathological Studies of the Nose and Sinuses in Allergy. *Jour. of Allergy*, 1:43-71, 1929.
14. Finck, H. P.: Tissue Changes in the Nasal Mucosa. *Laryngoscope*, 37:783-797, 1927.
15. Personal Communications (Dean, Proetz and Johnston).
16. Dean, L. W.: Recent Advances in Otolaryngology. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:952-962, 1929.
17. Hirsch, O.: Nasal Polypi—Their Origin and Its Solution. *Zentralblatt f. Hals, Nasen-u. Ohrenh.*, 9:745, 1927.

18. Hirsch, O.: Catarrhal Inflammation of the Nasal Accessory Sinuses and Its Diagnosis. *Laryngoscope*, 37:1-9, 1927.
19. Billroth, Th.: Über den Bau der Schleimhautpolypen. Berlin, 1855.
20. Hopmann, C. M.: Die Papillären Geschwülste der Nasenschleimhaut. *Virchow's Arch. f. Path., Anat. u. Physiol.*, 93:213-258, 1883.
21. Hopmann, C. M.: Über Nasenpolypen. *Monatschr. f. Ohrenheilk. u. Rhinol.*, 19:161-167, 230-236, 1885.
22. Hopmann, C. M.: Was ist man Berechtigt Nasenpolyp zu Nennen? *Monatschr. f. Ohrenheilk.*, 21:152-156, 1887.
23. Zuckerkandl, E.: Über Normal und Pathologische Anatomie der Nasenhöhle. Bd. I, 1882, Bd. II, 1893.
24. Uffenorde, W.: Erkrankungen des Siebbeines, 1907.
25. Uffenorde, W.: Die Verschiedenen Entzündungsformen der Nasennebenhöhlenschleimhaut. *Zeitschr. f. Ohrenheilk.*, 72:133, 1915.
26. Uffenorde, W.: Entstehungsweise und Rückfallneigung der Nasenpolypen. *Arch. f. Laryng.*, 33:512-530, 1920.
27. Woakes, Edw.: Necrosing Ethmoiditis and Mucous Polypi. *Lancet*, 1:619-620, 1885.
28. Hajek, M.: Über die Pathologischen Veränderungen der Siebeinknochen im Gefolge der Entzündlichen Schleimhauthypertrophie und der Nasenpolypen. *Arch. f. Laryng.*, 4:277-300, 1896.
29. Cholewa: Warum Recidiviren Nasenpolypen? *Monatschr. f. Ohrenh.*, 34:103-112, 1900.
30. Cordes, H.: Über die hyperplasie, die Polypöse Degeneration der Mittleren Muschel, die Nasenpolypen und Ihre Beziehungen zum knöchernen Theile des Siebbeines. *Arch. f. Laryng.*, 11:280-335, 1901.
31. Yonge, E. S.: Observations on the Determining Cause of the Formation of Nasal Polypi. *Jour. Laryng.*, 22:517-530, 1907.
32. Watson-Williams: Discussion of Paper by E. S. Yonge. *Jour. Laryng.*, 22:517-530, 1907.
33. Grünwald, J.: Lehre von den Naseneiterungen. München, I. Ed., 1893.
34. Alexander, A.: Die Nasenpolypen in Ihren Beziehungen zu den Empyemen der Nasennebenhöhlen. *Arch. f. Laryng.*, 5:324-381, 1896.
35. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses. (Trans. by Heitger and Hansel). C. V. Mosby Co., St. Louis, 1926.
36. Uffenorde, W.: Wie Entstehen die Retronasalpolypen? *Zeitschr. f. Hals-Nasen-Ohrenh.*, 6:134-152, 1923.
37. Fraser, R. S.: Nonsuppurative (Nasal) Sinusitis and the Principles of Hirsch. *Bull. Battle Creek San.*, 24:36-50, 1929.
38. Seifert, O., and Kahn, M.: Atlas der Histopathologie der Nase. J. F. Bergmann, Wiesbaden, 1895.
39. Lewy, B.: Die Beziehungen der Charcot-Leydenschen Krystalle zu den Eosinophilen Zellen. *Zeitschr. f. klin. Med.*, 40:59-83, 1900.
40. Jacobsthal, E.: Über Phagocytoseversuche mit Myeloblasten, Myelocyten und Eosinophilen Leucocyten. *Arch. f. Path., Anat. u. Physiol.*, 234:12-21, 1921.

41. Mullin, W. V., and Ball, R. P.: Studies of Pathologic Tissue Removed from Chronically Infected Nasal and Accessory Sinuses. *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:192-195, 1928.

42. Israel, S.: Chronic Disease of the Maxillary Sinus. *Arch. Oto-Laryng.*, 7:565-579, 1928.

43. Henke, F., and Lubarsch, O.: *Handbuch der Speziellen Pathologischen Anatomie und Histologie*. Vol. III. J. Springer, Berlin, 1928.

44. Leicher: Über Allergische Rhinitis (Ausschliesslich des Heuschnupfens). *Zeitschr. f. Hals-Nasen-Ohrenh.*, 20:238-250, 1928.

XXXVII.

LATENT MENINGITIS COMPLICATING SURGICAL
MASTOIDITIS: THE SIGNIFICANCE OF HEAD-
ACHE: LIMITATION OF THE PEDIATRI-
CIAN'S ATTENDANCE.*

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With a thin plate of bone separating the temporosphenoidal lobe from the tegmen antri, where infection is always found in mastoiditis, and a membrane on the promontory windows alone barring this infection from the internal ear with ready access to the brain, why isn't meningitis found more often as a complication of middle ear abscess from the very beginning? No answer to this question can be anything but speculative and theoretical, unless it be said that it is really existent under these circumstances very often, but is not recognized or does not advance beyond that condition which is known as a "localized meningitis," from which recovery can occur without the usual signs of meningitis. It would seem reasonable to suppose that an acute suppurative middle ear associated with a temperature varying from 100 to 102 F. every day, and a severe headache for a week would excite the suspicion of at least a meningeal irritation on the part of the attendant, as an uncomplicated otitis media rarely shows pain anywhere or fever after a free and early incision of the drum. However, despite such universally recognized facts, I have twice had patients turned over to me for treatment with this history over a two-week period. They were children, in the hands of a pediatrician of reputation and experience, but, to my amazement, the headache which had been constant he mentioned to me quite casually and seemed to think had little bearing on the case. In fact, I found out the nature of it and its persistence only by searching questioning of the family. Of course, any otologist

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

of experience dreads headache before or after a mastoid operation or during the course of an otitis media, unless otherwise accounted for, and even then I am never satisfied and never willing to accept the usual pediatric explanation for fever and headache, which is seemingly always: "gastrointestinal irregularity." I wish to call attention to the headache, generally frontal, which persists and is sufficiently bad to make the patient complain of it frequently, which is associated with an otitis media from the beginning or shortly after its beginning, especially when the infecting organism is the hemolytic streptococcus or any of the other unusually virulent bacteria. I am assuming that any other positive cause of headache has been ruled out, of course.

I have seen six cases of otitis media ending in mastoidectomy and death from a meningitis that certainly existed before operation and was not recognized at the time. I do not mean to say that recognition at operation would have prevented death, but I do say most emphatically that two of these deaths certainly and probably a third could have been avoided if the attendant (a pediatrician) had recognized the significance of the headache which had existed for a week before he turned the cases over to an otologist who did an immediate operation on the mastoid. Three of these cases were unhappily my own and three I saw in consultation, and not one of the deaths could be fairly chargeable to neglect or faulty operative technique on the part of the otologist. Of the six, five were under the complete control of the pediatrician, though, in all fairness, I will state that none of the usual signs of meningitis was present, though headache was in every case. Moreover, no signs appeared sooner than ten days after operation; in fact, three of them showed no localizing signs till four weeks thereafter, unless headache can be so regarded, as it did exist.

It seems pertinent here to point out the increasing tendency on the part of the internist, and especially the child specialist, to assume expert knowledge of the treatment of discharging ears, a procedure fraught with grave danger to the patient in the light of possible and frequently serious complications, among which may be mentioned not only death but chronic otorrhea, facial paralysis and deafness. With this in mind, I can see no reason why an internist or a child specialist should

feel competent to treat such a case, even though he were willing to assume the serious responsibility involved, always granting that an experienced otologist were obtainable. If a patient developed pneumonia while under treatment for laryngitis at the hands of a laryngologist, would any reputable throat specialist treat the patient further? Yet a pediatrician seems to feel perfectly competent to treat an otitis which develops in the course of a measles or coryza in a child, contrasting with the fact that pneumonia is a self-limited disease, and the patient generally recovers without any care other than rest and supportive treatment, and would be as safe in one man's hands as another's. The question thus arises: where does an otitis media cease to be pediatric and become otologic? The answer is painfully simple: when a complication arises. This is obviously not fair to the patient, nor, while of less importance, to the otologist.

In children fever from any source may readily cause headache at first, but fever and headache, when associated with an acute discharging ear, have a common origin and are not caused the one by the other when the discharge has lasted for as long as, say, five days or more. When such a picture is seen, especially when a leucocyte count as high as 10,000 exists, the possibility of meningeal irritation from exudate in the mastoid cells is to be always kept in mind.

Case 1.—Girl, 7 years of age; earache followed in two hours with spontaneous rupture of drum and watery discharge for a week; at the end of this period the pain and headache were still present. It was here that I first saw her and advised immediate operation. There was nothing unusual in the operation nor was there any exposure of the dura. The child returned to school in four weeks with a healed wound, but came home that same afternoon complaining of a severe headache. This the attendant and I thought was due to an obstinate constipation of five days' duration, though the fever was 99.5 F. After two days' efforts the bowels were moved and the fever, headache and lassitude all disappeared. She returned to school but was forced in two days to stay home, her headache and fever having returned. I now felt she had a meningitis and a lumbar puncture confirmed the diagnosis. She died in ten

days from the time of first (postoperative) headache, four weeks after the mastoid operation.

Case 2.—Boy, 12 years of age; very neurotic; earache at midnight; drum incised next morning; seemed very ill from the beginning, but parents hesitated to have an operation, as they thought an abscess in the ear of only five days' duration could hardly be provocative of a mastoid abscess. But the presence of the hemolytic streptococcus, the profuse watery discharge and the boy's great suffering made me insistent. After two days' delay, which I fear was fatal, the operation was done, disclosing a mastoid process full of dark blood, but no dura was exposed. After the routine gradual decline of fever, on the tenth day he complained of headache and his temperature began to show an evening rise to 101 and he felt chilly. The wound was reopened and a small necrotic area at the tip removed, and the jugular vein was ligated, after which the fever ran from 100 to 101 F. At the end of three weeks a lumbar puncture showed an advanced meningitis, and he died eight days after. Headache was a prominent symptom in the last ten days but was not constant. His marked neurosis made estimation of his subjective symptoms difficult.

Case 3.—Girl, 8 years of age. A week before I saw this patient the left drum had been incised and the right had ruptured spontaneously that night, two hours later. I found a profuse, pinkish discharge, an obviously ill child, tossing about the bed and complaining constantly of headache, which *subsequently* I found had been a prominent symptom for a week. I advised immediate operation but consented to wait till the next morning (twelve hours), when the operation revealed great destruction of the mastoid cells and a small exposure of the dura on the left side. She left the hospital in a week, apparently in good condition. Eleven days later (the eighteenth day after operation) she complained of great pain in the head, her fever rose to 102.5 F., and her leucocytes were 19,150. She was returned to the hospital and a ligation of the jugular vein done, as the signs were those of a phlebitis. No change being secured for the better, a lumbar puncture showed a meningitis, and the child died twenty days after the mastoid operation.

These three patients were repeatedly subjected to all the usual tests for determining the presence of a meningitis and never showed any reaction to the tests till the lumbar puncture was done. Headache was a prominent sign in every one of them, both before and after operation. It is almost certain that they all had a localized meningitis before operation, which remained walled off for some time (ten days to four weeks). The mastoidectomy apparently acted as a decompression operation and further delayed the appearance of symptoms till the ring of adhesions localizing the infection was broken through.

The three cases of meningitis I have seen in consultation are very similar and can be briefly summarized: Two were children and one an adult. Headache was seen in all, and no warning or sign of meningitis other than this was observed for a period of a month in the adult patient, and in the other two, three weeks and ten days, respectively. Delay in consenting to operation was certainly the cause of the meningitis in the last one.

CONCLUSION.

1. Persistent headache, especially frontal, associated with an otitis media, of any period of five days or more, to be regarded as a sign of possible meningitis.

2. Absence of signs of meningitis in these cases before operation probably due to localization of process; absence after operation probably due to decompression effect of mastoidectomy.

3. Cases of otitis media strictly the province of the ear specialist, though there is no valid objection to the opening of a drum by the pediatrician in order to spare the patient the added pain and danger of delay that might ensue by waiting several hours to secure the services of an otologist. After the myringotomy, however, the patient should be turned over to the ear man for treatment.

4. Exposure of dura at operation, either by disease or by the surgeon's instrument, bears no relation to cause of meningitis in such cases as touched upon in this paper. In fact, I have never seen a case produced by such exposure alone (*i. e.*, unless dura wounded at the time). At least 75 per cent of the

mastoidectomies that I have done showed such exposure and I have never seen any untoward result that could be attributed to it.

5. Hemolytic streptococcus found in all six cases. This organism will be more often found, in my belief, than any other in such cases as those described by at least the frequency represented by 85 per cent.

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XXXVIII.

CLOSURE OF ALVEOLAR FISTULA BY SCAR TISSUE FORMATION.*

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In searching the literature little mention of any procedure for closing off the opening from the mouth into the maxillary sinus is found.

Alveolar fistule into the antrum are the result of four different conditions, according to Lyons.¹

1. Dental roots lying within the antrum, which cause a fistula when extracted. Such a fistula, with the resulting antrum infection, is comparatively rare and, of course, unavoidable.

2. Roots extending to, but not through, the floor of the antrum, where after extraction the use of curets or probes accidentally penetrate the antrum and result in a fistula.

3. Dental apical abscesses or granulomas breaking down the floor of the antrum, resulting in fistula after extraction of the tooth.

4. An accident occurring during extraction, when the root is forced into the antrum but where the dental root does not involve the floor of the antrum.

It is best to assume that a fistula has resulted and that the antrum is infected when pus is found draining from the alveolar pocket a day or two after extraction of a tooth. If such is determined to be a fact, the proper treatment is to make a window resection of the nasal wall of the antrum through the inferior meatus. It will be sufficient. This is not the type of case with which this paper deals.

When a fistula has been present a month or longer it becomes lined with epithelium, thereby making a permanent opening and one that curettement will not suffice to eliminate. In the treatment of a chronic fistula it is first necessary to

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

relieve the chronic infection of the antrum, and secondly, to eliminate the fistula. When one is positive that the antrum is no longer infected the fistula may then be closed. Simple suture will suffice if the fistulous opening is not too large, and the soft tissues approximate without tension, but if the tract is large, a plastic closure may be made.

J. Parsons Schafer² states that after the examination of hundreds of skulls he is convinced that the second molar tooth protrudes through the alveolar process into the antrum and is the cause of infections of the antrum more than is generally supposed. Drury³ has never seen a case of fistula developing from the extraction of any tooth except the second molar. It must not be forgotten that any tooth, from the central incisor back, may be the cause of maxillary sinus infection and alveolar fistula. Frequently patients have had a tooth removed and so much curettage has been done around the root opening that a large hole was broken through the alveolar process.

Hempstead⁴ reports sixty-three cases where there was a fistula from the antrum through the alveolar process out of 385 chronic maxillary sinusitis cases.

Shearer⁵ writes: "It has been my experience to see in many instances not only infection of a normal maxillary sinus but even the loss of a tooth into the antrum from attempted removal of these teeth with simple extraction and blind curettement."

Novitzky⁶ says that he has found many dead teeth draining into the antral membrane without perforating it.

Dunning⁷ mentions that it has been proven clinically that a permanent opening from the mouth into the antrum may produce an ascending infection into the maxillary sinus; yet, how often do we see the antrum, in these chronic cases of oral origin, irrigated for weeks through the opening in the mouth and packed with gauze.

Why should a large alveolar opening into the antrum be permitted to exist? This question is answered in many ways by different authors.

Thomson⁸ writes: "Although subject to exacerbations after contracting an acute catarrh, patients frequently prefer to tolerate the inconvenience and wear an obturator indefinitely,

rather than submit to operation." I am certain that our American patients would not.

Hajek⁹ has observed cases of maxillary antrum suppuration which were irrigated for years through alveolar fistulas and which had to be radically operated upon. Most of the irreparable changes are found in the region of the peg of the prosthesis, which in itself makes healing impossible. Many operators have attempted by extensive resection of one or two alveolar sockets to produce a free communication which would remain open and maintain drainage. This is, however, not a desirable procedure, since with every inspiration the contents of the mouth will be drawn into the maxillary antrum, while with every forceful expiration, as in blowing the nose, the contents of the maxillary antrum will enter the mouth cavity.

The presence of an open canal is as unpleasant as it is injurious on account of the annoying and disgusting taste of pus and the aspiration of food particles into the maxillary antrum by forced inspiration through the nose.

Phillips¹⁰ reports that one disadvantage of an opening through a tooth socket is the necessity of wearing a dental prosthesis; another disadvantage is that the opening through the process requires constant care to prevent infection from the mouth by aspiration of buccal secretion into the antral cavity. No mention is made about how to close these fistulas.

Denker¹¹ states that these fistula openings close themselves by granulation tissue. Some may do so, but he does not mention how to close those that do not.

Alfred Bruck¹² writes in his book that it is difficult to keep the small fistula open and that they heal promptly. No American rhinologist would attempt to keep them open, but he would do everything he could to get them to close.

Brown¹³ writes that by closure of the alveolar opening we cut off a fertile source of infection from below; and by performing a proper intranasal operation we obtain and maintain, permanently if necessary, as is needed in chronic cases, an opening into the antrum whereby we are assured ease of medication and free aeration, which, to my mind, is of utmost importance in all infections of this sinus, and is impossible to obtain where intervention is undertaken through the oral cavity.

Lyons¹¹ reports, in the treatment of chronic fistula, "First, to relieve chronic infection of the antrum, and secondly, to eliminate the fistula and its adjoint pathology." "When one is positive that the antrum is no longer infected, the fistula may then be closed."

In case of a permanent opening between the maxillary sinus and tooth socket, Mead¹⁵ advises closing the opening by making an incision and turning back a flap of gum, excising the bone and bringing the edges of the wound together and suturing.

Dean¹⁶ dissects out the ingrowing epithelium, removes sufficient of the process so that the flaps of mucous membrane will come together and fastens his sutures over rubber so that they will not cut through. He reported twenty-three cases closed in this way.

Hempstead¹ does not use packing in the cavity, and further treatment is carried on by dry suction. Irrigation is never resorted to, as it causes a water-logged condition of the mucous membrane, which is unfavorable to healing. Large fistulas often close after the described treatment, but if they do not they are closed by elevating the edges and sliding a flap of periosteum and mucous membrane from the palatal side, as advocated by Gardner.

Welty,¹⁷ under general anesthesia, made an incision at each end of the fistula, on outer side of the alveolus, and carried well up to where bone had been removed in the antrum operation. Periosteum and mucous membrane were removed from the alveolus, and afterward the whole of the outer border of the alveolus is removed into the antrum. A similar incision was made on the inside at each end of the fistula, periosteum and mucous membrane elevated from the bone, and the bone removed. An incision was now made in the median line of the hard palate its entire length and the periosteum elevated over the entire area. It is easy to understand that by this procedure all tension will be removed when the sutures are introduced. That the sutures may not cut the tissue, two perforated lead discs are used on each side and the sutures are tied rather snugly. This can remain for ten days.

Welty: "I know there are many persons about the country suffering from a fistulous communication with the antrum of

Highmore, who can be readily cared for in the way I described. However, I must insist that the radical operation for the antrum of Highmore be performed."

Beck¹⁸ remarks that there is nothing in plastic surgery that gives as much trouble as the closing of an opening as described by Dr. Welty, and that if he can close it by this radical method he has added a great deal to our technic.

Drury³ advocates, first, sterilization of the fistulous tract with mercurochrome; then, introducing closure by application of liquor epispastius (cantharides liniment), care being taken not to touch the neighboring parts to produce unnecessary blistering. Frequency of application depends on the reaction obtained, but usually, with the treatment repeated daily, the aperture gradually closes.

Maybaum¹⁹ reports that an alveolar sinus of small caliber responds readily to treatment by the use of cantherizing agents, provided, however, the antrum infection has been eliminated. The problem of obliterating a sinus of larger size is a more difficult one.

Despin²⁰ closes these fistulas by using a saturated solution of phenic acid.

Plastic surgery has failed to cure many of these fistulous openings. There are several reasons for this. Among them, first, is the failure to cure the antrum infection before attempting to close the fistula; second, postoperative infection; third, improper technic and the lack of means for preventing the sutures to cut through. In many of these cases, where plastic surgery has failed, and in other cases where surgery of the fistulous tract was not allowed or tried, the fistula may be closed by stimulation of the formation of scar tissue, until the entire opening closes by the contraction of the newly formed scar. By this method I have closed small fistula, also fistula so large that an index finger could be inserted into the opening.

The method used in cases was the cure of the antrum infection, either by doing the radical antrum or the intranasal drainage through the inferior meatus. It was not necessary to do a radical operation in all cases, because some were cured by the simpler intranasal operation, plus suction. Then the lining epithelium of the fistulous tract was destroyed, using the electric cautery; the more reaction following the burning, the

quicker the closure. After this the use of trichloroacetic acid, applied within the tract once or twice a week, until the tract closed by scar tissue.

This method of closure requires considerable time, but when the object is explained no patient refused the treatment.

The following cases had alveolar fistula that failed to close by themselves; however, when the above method was used, the result was most gratifying.

CASE REPORTS.

White, male, 40 years of age, who had all upper teeth extracted three months before. Since then has had droppings into the throat. Had a purulent discharge into the mouth. Considerable pain over the right side of the face.

Examination of nose: Large spur low down on right side of septum. Transillumination shows right antrum to be dark; all other sinuses clear. All teeth have been extracted. There is a discharging sinus, diameter of slate pencil, in root socket of upper right first molar. Puncture and washing of right maxillary antrum; water returned with yellow, foul smelling pus.

Operation: Large opening made into antrum through the inferior meatus. Postoperative treatment: Irrigation with water, suction and spraying 2 per cent mercurochrome into antrum. When antrum was well, the fistulous opening was cauterized with the electric cautery, and cauterized eight times thereafter with trichloroacetic acid, when the newly formed scar tissue closed the fistulous tract.

Mr. D. F. C., age 47, white, male. Nine months before, the patient had upper right wisdom tooth pulled, which was followed by pain and a tight sensation over right cheek. Dentist washed antrum through the tooth socket, after which he got relief from pain. He has had a bad taste in the mouth ever since.

Examination: Sinus the size of a lead pencil in tooth socket of upper right wisdom tooth, draining purulent mucus. Right maxillary antrum transilluminates dark. Puncture and washing of right antrum through inferior meatus; water returned with thick, foul smelling pus.

Operation: Right inferior turbinate fractured upwards. Large opening made into antrum in inferior meatus, using a chisel and biting forceps. Turbinate replaced. Daily irrigation and spraying with mercurochrome, 2 per cent. As soon as the antrum was clear the fistulous tract was cauterized with electric cautery, and cauterized at four day intervals thereafter with trichloracetic acid for ten times, when the newly formed scar tissue completely closed the tract.

Miss K., white, female, age 38. One year before, the patient had upper left second molar tooth extracted. This was followed by pain in the left cheek and purulent discharge into the mouth. Dentist curetted the tooth socket several times, attempting to get it to heal, but instead it became larger.

Examination: Large fistulous tract in region of upper left second molar socket. A little finger could be inserted into the tract. Left antrum transilluminates very dark. Puncture and washing of maxillary fossa: much thick pus came with washings.

Operation: Under ether anesthesia, a radical antrum operation was performed and a large opening made in inferior meatus under inferior turbinate, leaving turbinate in place. As soon as antrum was clean, the fistulous tract was cauterized with electric cautery, and following that twice a week cauterized with trichloracetic acid, over a period of three months, when the large tract finally filled in with scar tissue and remained tightly closed.

Mrs. F. D., white, female, aged 34, one year ago had all teeth removed and, because of pain in the left face, the dentist opened into the left antrum through the tooth socket. He irrigated the sinus through the mouth for a number of weeks, after which a large opening was left from which so much pus discharged that she could not wear her upper plate.

Examination revealed a large fistula in the upper left second molar socket one-half inch long and one-fourth inch wide; this was draining pus.

Operation: Under local anesthesia, anterior end of left inferior turbinate removed. Large opening made in region of inferior meatus into antrum. When the antrum was clean the fistulous tract was cauterized with electric cautery and then cauterized with trichloracetic acid, eight times, over a period

of two months, when the scar completely closed the sinus, after which the patient could wear her upper plate with comfort.

Mr. H. S., age 52 years, had upper teeth removed one year ago. Since then has had pain in right cheek, with a discharging sinus in a tooth socket on the right side.

Examination shows a fistula the size of a slate pencil in the region of the upper right molar tooth socket, discharging thick pus. Right antrum transilluminates dark. Puncture and washing; water returned with much thick, yellow pus.

Operation: Radical antrum operation right side, with large opening in inferior meatus, leaving the inferior turbinate intact. Antrum was full of polyps and pus. Alveolar fistula closed promptly with scar tissue after having been cauterized with trichloroacetic acid twice a week for ten times.

Mrs. W. J. R., four years ago had upper teeth removed. Since then has had a discharging alveolar sinus on right side, which a dentist has been washing at times since then. She cannot wear her plate because of the secretion under it.

Examination shows a discharging sinus, about one-fourth inch in diameter in region of upper right second molar socket. Right antrum transilluminates dark. Puncture and washing of right antrum through inferior meatus; water returned with thick, yellow pus.

Operation: Anterior one-fourth of right inferior turbinate removed. Large opening made through inferior meatus into antrum. As soon as antrum was clean, the alveolar sinus was cauterized with trichloroacetic acid four times at weekly intervals, when the sinus closed by scar tissue formation.

REFERENCES.

- 1-14. Lyons, Horace R.: "The Treatment of Alveolar Fistula Secondary to the Extraction of Teeth." *Transactions of Am. Laryngol., Rhinol. and Otol. Society*, 1925, page 81.
2. Schafer, Parson, Jr.: "The Nose and Olfactory Organs," 1920, page 114.
3. Drury, Dona W.: *Boston M. and S. J.*, 191:1123 (Dec.), 1924.
4. Hempstead, Bert E.: "Intranasal Surgical Treatment of Chronic Maxillary Sinusitis." *Archives of Otolaryngology*, Nov., 1927, page 426.
5. Shearer, Wm. L.: "Pathology of Alveolus—Relation to Maxillary Sinus—Method of Approach." *Transactions of the American Laryngol., Rhinol. and Otol. Society*, 1927, page 558.

6. Novitsky, J.: "Dead Teeth and Antral Pathology." Illinois M. J., Oak Park, 1920, XXXVIII, 143.
7. Dunning, Henry Sage: "Maxillary Sinusitis of Oral Origin." Laryngoscope, Oct., 1925, page 766.
8. Thomson: "Diseases of Nose and Throat." 1926, page 280.
9. Hajek: "Nasal Accessory Sinuses." Vol. I, page 249, page 288.
10. Phillips: "Diseases of Ear, Nose and Throat," page 579.
11. Denker, A.: "Die Krankheiten der Nase und ihr Nebenhohlen," page 320.
12. Bruck, Alfred: "Die Krankheiten der Nase und Mundhohle," page 121.
13. Brown, Louis E.: "Relationship of Teeth to Pathology of the Maxillary Sinus." ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1925, page 150.
15. Mead: "Diseases of the Mouth," page 310.
16. Dean, L. W.: "A Method of Closure, a Sinus Between the Antrum of Highmore and Mouth." J. Am. A., 1913, LXI, 1613.
17. Welty, Cullen F.: "Closure of Fistulous Openings Through the Alveolar Process Into the Antrum of Highmore." Transactions of Section of Laryngology, A. M. A., 1920, page 198.
18. Beck, Joseph C.: Discussion of Welty's paper, p. 200.
19. Maybaum, J. L.: "Two Cases of Empyema of Maxillary Sinus of Dental Origin—Alveolar Fistula, Radical and Plastic Operation." ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, March, 1923, page 273.
20. Despin: "Treatment of Dental Fistula." J. de Med. de Bordeaux, 92, 405, July 25, 1921.

XXXIX.

STUDIES ON THE ACCESSORY NASAL SINUSES.

THE COMPARATIVE MORPHOLOGY OF THE NASAL CAVITIES
OF AMPHIBIA.

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ST. LOUIS.

In a series of investigations, of which this study is the first, an attempt was made to correlate the findings of earlier workers with our own in order to trace the phylogenetic history of the maxillary sinuses. However, a thorough survey of the zoologic and anatomic literature disclosed the amazing fact that the comparative morphology of the maxillary sinuses has been more or less slighted. It seemed apparent, therefore, that further studies on the morphology of the nasal cavities in representatives of the various vertebrate groups were necessary and essential in properly orienting the workers in animal experimentation in the field of otolaryngology.

Among the teleosts, ganoids, elasmobranchs, and groups lower in the classification of vertebrates, respiration is taken care of by gills. The olfactory organs are not accessory structures in the function of respiration. They consist of a paired sac-like structure, more or less completely enclosed within a cartilaginous or bony investment on the upper surface of the snout. These olfactory sacs communicate with the exterior through an opening, the external nares, which in turn are divided by a fold of skin into two openings, the anterior and posterior nares, which have no communication with the buccal cavity. However, the oronasal groove of the elasmobranchs is suggestive of such a communication.

The mucous membrane of these structures is raised in a series of folds and, as described by Blaue,¹ is comprised of sensory and ciliated epithelium.

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The dipnoan fishes, possessing a dual set of respiratory structures, namely, gills and lungs, are the first order of vertebrates that show a communication of the olfactory organ with the oral cavity. During the period when the lungs are used for respiration, the olfactory sac serves as a respiratory passage. Structurally, the olfactory sac, enclosed by a cartilaginous trellis work, closely resembles that of elasmobranchs.

For the convenience of the reader, a brief classification of the amphibia, taken from Parker and Haswell,² is included here.

ORDER I. URODELA.

(a) *Perennibranchiata*.—Those forms that retain gills throughout life. *Proteus*, *necturus* and *siren* are examples of this group.

(b) *Derotremata*.—Gills are lost in the adult stage but retain a gill cleft. *Amphiuma* and *cryptobranchus* are examples of this group.

(c) *Myctodera*.—In the adult the gills and gill cleft are lost. *Salamander* and *triton* represent this group.

ORDER II. ANURA.

This order has no tail, but four legs in the adult. Represented by frogs and toads.

ORDER III. GYMNOPTIONA.

No legs in this group. Represented by limbless caecilians.

The fourth order, the *stegocephali*, are an extinct species and are probably the precursors of the above mentioned orders.

All representatives of each specimen of amphibia were fixed in Zenker's fluid, the heads were then decalcified, run through celloidon, and cut in serial transverse sections.

URODELES.

Adult forms of *Necturus*, a representative of the *perennibranchiate* type of amphibian, were selected for this study, the species being *necturus maculatus*. This amphibian spends most of its existence in water.

The nasal cavities have a simple structure, and are in the form of a long tube. The external openings are on the upper anterior end of the snout, while the internal openings, or

choanae, are located several centimeters posteriorly and communicate with the oral cavity. Both external and internal openings have the form of a cleft.

The epithelium at the external and internal openings is respiratory in type, stratified, with the upper layer somewhat flattened. This type of epithelium extends inwardly and becomes interspersed with olfactory epithelium, the arrangement of which is arranged in buds. There is no continuous layer of respiratory epithelium from the external to the internal opening. The olfactory epithelium consists of a clear zone at the surface, while lower down one finds olfactory cells with round nuclei and supporting cells with oval nuclei. No cilia were found in this genera. The structure of the olfactory epithelium resembles that of fishes and *Proteus*, of which a detailed study has been made. (Blaue.¹) The olfactory epithelium surrounds the nasal cavity on all sides in the central part of the nasal cavity, and at places these olfactory buds have a greater development and form blind pouches of various shapes and sizes. According to Seydel, these structures may be the forerunners of Jacobson's organ. No evidence was found of a nasolacrimal duct, sinus, or glands supplying the nasal mucosa. The palate is a primitive type, for a hard palate is not found in amphibians. The nasal passage opens directly into the buccal cavity.

Seydel² studied two members of the perennibranchiates, namely, a species of *proteus*, which has a nasal structure very similar to *necturus*, and *siren lacertina*, which shows a more complicated structure than either *proteus* or *necturus*. In *siren*, respiratory epithelium lines the floor and part of the medial wall throughout the length of the nasal cavity, while the remainder is covered with olfactory epithelium arranged in buds. On the medial floor of the *apertura nasalis interna*, and beginning at the lateral corner of the olfactory sac, a flat channel-like depression goes obliquely, anteriorly and medially. Its posterior end is shallow and lies in the region of respiratory epithelium; anteriorly it gradually deepens into the border between *regio olfactoria* and *regio respiratoria*. Its anterior end is in the form of a large blind sac. The medial part of this sac is lined with sensory epithelium which is not bud shaped. The lateral part of this sac is lined with tall cylin-

drical epithelium which is respiratory in type. According to Seydel, this blind sac is Jacobson's organ. A well developed gland empties on the floor between the medial and lateral portions. In this case, Jacobson's organ has no cartilaginous support.

Another class of the urodel group is the derotremata, which is represented in this study by an adult *Amphiuma tridactylum*. *Amphiuma* has a rather simple arrangement of the nasal cavity, not much more developed than *necturus*. The external and internal openings have the shape of a cleft, and the nasal cavities are tube shaped with a lateral outpouching of approximately the central two-thirds of its length.

The nasal cavities have a complete cartilaginous framework. The epithelium is of two types, respiratory and olfactory. The respiratory type (stratified) is present exclusively at the external and internal openings. As one goes posteriorly from the external opening one sees the olfactory type (buds) appearing first medially and then also dorsally and partly on the ventral side. The outer lateral side continues as respiratory epithelium throughout the length of the nasal cavity. The olfactory type is arranged in buds. The olfactory epithelium is surrounded by a well developed glandular system. The lateral outpouching has olfactory epithelium superiorly and laterally, which may be a rudimentary Jacobson's organ. No cilia or a nasolacrimal duct were seen. As one proceeds posteriorly, the lateral pouch disappears and the olfactory epithelium decreases, remaining longest on the medial side. Wilder⁴ made a study of the nasal region of *amphiuma*, and gives a detailed study of the nerves and cartilaginous and bony skeleton of the head.

According to his illustrations, the lateral pouch of the nasal cavity does not contain olfactory epithelium as was found here. Wilder considers this as a sort of maxillary sinus. He also describes a small lateral pouch of the nasal cavity of *Menopoma Allighaniense*, which apparently is lined with olfactory epithelium.

However, the writer believes it is a very primitive form of Jacobson's organ on account of the presence of sensory epithelium. Wilder⁵ studied the lateral nasal gland which empties near the external opening, and considered its function as a

moistening and cleansing organ. Besides this lateral gland there is a well developed gland medial and superior to the nasal cavity.

An adult salamander, *Plethodon Glutinosus*, a member of the myctodera, another branch of the urodeles, was selected for this work. The nasal cavities of this amphibian show a greater development than the previously mentioned species. The anterior end has a small round opening which leads into a rather large dome-like space or vestibule. There is a small pouch laterally close to the anterior end and lined with respiratory epithelium (cylindrical).

This pouch receives the nasolacrimal duct. The olfactory epithelium is not arranged in bud formation, instead, the supporting and olfactory nuclei are spread over greater areas. At the anterior end, olfactory epithelium lines almost the entire superior, medial and ventral aspect of the nasal cavity, while laterally respiratory epithelium is present. Both types of epithelium are ciliated. Passing posteriorly to the entrance of the nasolacrimal duct there is a narrowing of the lateral extension. Laterally one sees a tube which ends blindly anteriorly and is lined with ciliated olfactory epithelium. This proceeds posteriorly and empties into the lateral end of the nasal cavity, which has extended more laterally again. The olfactory epithelium lines the lateral end of the nasal cavity for a short interval and then is replaced with respiratory epithelium. The blind duct is perhaps Jacobson's organ. Mihalkovics⁶ designates the lateral recess as a maxillary sinus, while Seydel designates it as Jacobson's organ. Neither mentioned a blind duct in their description. The lateral recess of the nasal cavity, after it is lined with respiratory epithelium, is probably a maxillary sinus, the writer agreeing with Mihalkovics on this point. The nasal cavity continues in this form to the internal aperture, which opens in a slit arranged diagonally. The olfactory epithelium disappears next on the medial surface. The respiratory epithelium, especially in the lateral recess, has goblet cells. There is a well developed interseptal gland. A few glandular structures placed dorsally and ventrally to the nasal cavity are present. No glands were found laterally, and also none seen around the blind duct, which is well surrounded with bone and cartilage. The salamander has

a complete cartilaginous and bony framework for the nasal cavity. The maxilla has a medial process which is a hint of the formation of a secondary palate.

GYMNOPHIONA.

Gymnophiona are another of the family of amphibians. The writer did not have the opportunity to study this family, since they do not exist in North America. In describing the nasal cavity of this genera, Seydel³ quotes P. and F. Sarasin and also Wiedersheim. The nasal cavity of *Ichtyopsis* is broad and roomy, and is more or less divided into an olfactory and respiratory portion by a turbinate-like structure on the floor of the nasal cavity, which extends along the entire length of the floor. The olfactory portion is medial and is lined with olfactory epithelium, while the lateral respiratory portion is lined with respiratory type of epithelium. Jacobson's organ is situated as a flattened channel beneath the respiratory duct and is lined with olfactory epithelium except on the upper and lateral surfaces. It receives the nasolacrimal duct in its lateral anterior portion, according to the description of P. and F. Sarasin. Jacobson's organ terminates in the nasal cavity close to the choanæ; the choanæ are located posteriorly to the respiratory passage of the nasal cavity. Numerous glands terminated in Jacobson's organ at the border of the olfactory and respiratory epithelium. According to Wiedersheim, several other members of *Gymnophiona* (*Epicrion*, *Coecelia*) also have Jacobson's organ as a closed blind sac lying beneath the nasal cavity and emptying at the choanæ. Jacobson's organ in the *Gymnophiona* is unique. The termination of the nasolacrimal duct into it finds no homology in the lower or higher forms of vertebrates. In reference to the development of the nasal cavity, the *Gymnophiona* are more highly developed than the other amphibians. In other ways, however, they seem to be a primitive form of amphibian, absence of limbs, etc.

ANURA.

Adult forms of frogs were studied as representatives of the anura, the species being *Rana pipiens*. The nasal cavities have a more complicated structure than that of the urodeles, which is also true of other organs of the frog. The external nasal

openings are round structures located on the dorsal lateral part of the snout, and enter the nasal cavity on its dorsal surface, since the nasal cavity extends somewhat anteriorly to the external nares. A serial frontal section series was made, and the nasal cavities were studied from their anterior end to the choana.

At the most anterior portion one sees two blind cavities, also a thick cartilaginous nasal skeleton having a thick septal cartilage, with two lateral extensions, the superior and inferior paraseptal cartilages. These divide the nasal cavity into two parts. The upper cavity, according to Gaupp,⁷ is the *cavum medium*, while the lower is named the *recessus medialis cavi inferioris*, but the writer will designate this latter as Jacobson's organ. The upper cavity is lined with respiratory epithelium, while Jacobson's organ is lined with olfactory epithelium. Further posteriorly the external nares open into the *cavum principal*, which is round and lined on all sides except the lateral with olfactory epithelium. Laterally it is lined with respiratory type of epithelium. The *cavum medium* has not changed much except being somewhat larger, while Jacobson's organ has now united with the cavity *cavum inferioris*. Medially olfactory epithelium is present; laterally one sees the respiratory type. The cavities are still enclosed in a cartilaginous skeleton; the septal cartilage becomes thinner as one goes posteriorly. Somewhat posterior to this one sees a union of the *cavum medium* and *cavum inferioris* at their medial portion. The olfactory epithelium of Jacobson's organ does not take part in this but is seen as a round tube medial to the junction of the two cavities. The *cavum principal* sends a process (*infundibulum*) towards the lateral aspect of the *cavum medium* and the next development is the union of the *cavum principal* with the *cavum medium*. The *cavum inferior* has extended laterally and inferiorly to the maxilla. Further posteriorly the *cavum medium* and *cavum inferioris* fuse so that the *cavum principal* connects directly with the *cavum inferioris*. One sees the appearance of an elevation on the floor of the principal cavity—the *eminentia olfactoria*—and there is also a turbinate-like structure extending into the nasal cavity at about its central part, the *plica isthmi*. This divides the nasal cavity somewhat into a lateral and medial

portion, while the passage just below this is called the isthmus. Ciliated olfactory epithelium is present in the dorsal, medial and ventral surfaces of the *cavum principal*; the remainder is lined with ciliated respiratory epithelium. Dogiel⁸ made a detailed study of the olfactory epithelium of the frog, and those interested are referred to his article. The internal opening (*choana*) appears as a diagonal slit at the posterior end of the nasal cavity. There is a hint of the formation of a secondary hard palate in that the maxilla has a short medial process. The olfactory epithelium is supplied with mucous glands, Jacobson's organ being supplied with a well developed medial nasal gland, while the olfactory epithelium of the *cavum principal* has the usual type of Bowman's glands. A lateral nasal gland is also present, which empties into the *cavum principal*.

The physiology of the nasal cavity of the frog is interesting, for, according to Gaupp⁷ and others, the nose of the frog serves for respiratory and olfactory purposes. Also the blind cavities help to maintain the buoyancy of the frog in water. Experiments were cited that water does not enter the nasal cavity, for the cilia would be damaged if it should. Apparently the olfactory epithelium of the *cavum principal* tests the ingoing stream of air, while Jacobson's organ receives only the expiratory air current (perhaps to test the contents of the oral cavity), by way of the maxillary sinus. The frog can close the external nostrils by means of a system of muscles at the orifice. After air reaches the pharynx it is swallowed and delivered to the lung. Since the frog has no diaphragm, the abdominal muscles force the air out of the lungs.

REVIEW OF THE NASAL CAVITY OF AMPHIBIANS AND RELATED STRUCTURES.

The nasal cavities of *Necturus* and *Proteus*, the most primitive type of amphibians, are simple tubes, while the next development is either a sinking in of the floor (*siren*) or an extension, laterally, *amphiuma*, salamander, etc. Apparently the first purpose of the nasal cavity was olfactory, but in higher forms there results a respiratory function which is added. The greatest differentiation in the amphibia is in the respiratory area. Jacobson's organ seems to be a specialized form of

the olfactory apparatus. True turbinates are not found in the amphibia. Gegenbauer considered a turbinate a simple projection of bony skeleton of the wall of the nasal cavity, and in this regard the turbinates of reptiles and birds also fail to meet this definition. There the turbinates have cartilaginous support. In the frog there is a cartilaginous support of the olfactory eminence and plica isthmi which serve to increase the surface area of the nasal cavity and to divide somewhat the nasal cavity. They can be regarded as a beginning of pseudoturbinate formation, homologous to the turbinates of mammals. The nasolacrimal duct in the higher amphibians, salamander and frog, appears and enters a lateral pouch. In *Gymnophiona* the nasolacrimal duct enters Jacobson's organ.

There is no true maxillary sinus in the amphibia, because the maxillary bone has no hollow space. Neither is there a true hard palate; the maxillary bones do not send palatal process to meet the opposite side. The vomer serves as a bony covering of the oral cavity.

Discussion of the so-called maxillary sinus in the higher amphibia involves a discussion of Jacobson's organ also. According to Bawden,⁹ the most primitive position for Jacobson's organ is laterally. According to this view, *Gymnophiona* and the salamander have the most primitive location for this organ, while the presence of Jacobson's organ in *Amphiuma* is questionable. Seydel asserts that the lateral position of Jacobson's organ is only apparent, that embryologically this anlage is formed medially and Jacobson's organ attains a lateral position by means of a turning of the nasal sac. Seydel believes that it is possible that the maxillary sinus of mammals originated in Jacobson's organ of amphibia; however, this matter is controversial.

Mihalkovics does not believe Jacobson's organ exists on the lateral side of the nasal cavity of any animal and denies the existence of Jacobson's organ in the salamander. He states that in the formation of the nasal cavity the sensory type of epithelium first lines the entire nasal cavity, while later the respiratory type appears. He attributes the olfactory epithelium present on the lateral side of the nasal cavity of the salamander as an embryonic rest in the maxillary sinus.

He describes branches of the dorsal as well as the ventral branches of the olfactory nerve leading to these cells. Jacobson's organ in the higher vertebrates receives only a ventral branch of the olfactory nerve.

Neither Mihalkovics nor Seydel found a blind cavity in the salamander as found in the writer's specimen. Mihalkovics disagrees with Seydel as to the terminology of the organ in the floor of the nasal cavity of Siren and calls the lateral portion the maxillary sinus and only the medial side with olfactory cells Jacobson's organ. In regard to *Gymnophiona*, Jacobson's organ is cut off from the nasal cavity and empties into it by means of a small opening near the choana. It has the appearance of a sinus. However, it is not enclosed within bone.

The writer agrees with Mihalkovics in the terminology of the frog's nasal cavity. Seydel called the entire anterior portion of the *cavum inferior* Jacobson's organ, while the writer considers Jacobson's organ in the frog as a blind sac communicating with the *cavum inferior*.

The function of Jacobson's organ in the amphibia probably serves as an olfactory control of the contents of the oral cavity, while the so-called maxillary sinus serves as a passage for the conduction of the expiratory air current. The glands supplying Jacobson's organ probably serve to wash away foreign bodies with their secretion. Moreover, this secretion may be necessary for the olfactory function, although the function of Jacobson's organ up to the present time has not been settled definitely. Hamlin found that an increase in blood pressure caused an expulsion of the contents of Jacobson's organ in rabbits, and fall of blood pressure caused a sucking in of air.

Further studies on the comparative anatomy of the nasal structures of higher vertebrates are now under way and will be reported by the author in the near future.

BIBLIOGRAPHY.

1. Blaue, J., 1884: Untersuchungen über den Bau der Nasenschleimhaut bei Fischen und Amphibien, namentlich über Endknospen als Endapparate des Nervus Olfactorius. *Archiv für Anatomie und Entwicklungsgeschichte*, page 231, Jahrgang 1884.

2. Parker and Haswell: A Textbook of Zoölogy. Macmillan Co., 1921.
3. Seydel, O., 1895: Über die Nasenhöhle und das Jacobsonche Organ der Amphibien. *Morphologische Jahrbücher*, 23, page 453.
4. Wilder, H. H., 1892: Die Nasengegend von *Menopoma Alleghaniense* und *Amphiuma Tridactylum*. *Zoologische Jahrbücher*, Bd. 5, pp. 155-176.
5. Wilder, I., 1909: The Later Nasal Gland of *Amphiuma*. *Journal of Morphology*, Vol. 20, pp. 145-170.
6. Mihalkovics, V., 1899: Nasenhöhle und Jacobson's Organ. *Anatomische Hefte*, Bd. 11, pp. 1-108.
7. Gaupp, E. A.: Eckers und R. Wiedersheim's Anatomie des Frosches. 1904, Vol. 3, Friederich Vieweg und Sohn, Braunschweig.
8. Dogiel, A., 1887: Über den Bau des Geruchorgans bei Ganoids, Knochenfischen und Amphibien. *Archiv für Mikroskopische Anatomie*, Bd. 29, pp. 74-136.
9. Bawden, H.: The Nose and Jacobson's Organ with Special Reference to the Amphibia. *Journal of Comparative Neurology*, 1894, p. 115.
10. Hamlin, H., 1929: Working Mechanisms for Liquid and Gaseous Intake and Output of Jacobson's Organ. *American Journal of Physiology*, 91, pp. 201-205.

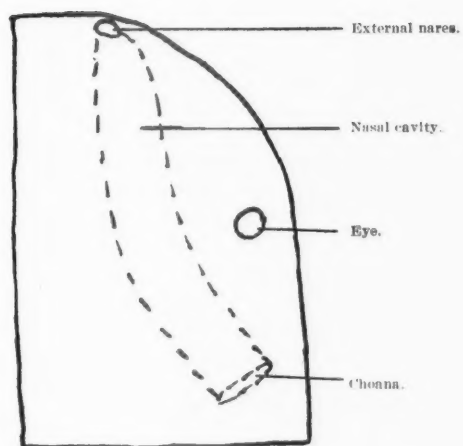


Fig. 1. *Nectures maculatus*. Diagrammatic dorsal view.

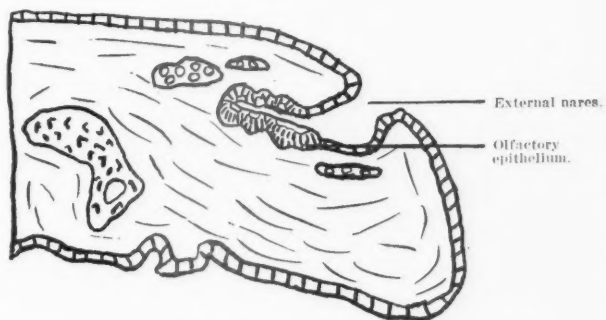


Fig. 2. *Nectures maculatus*.

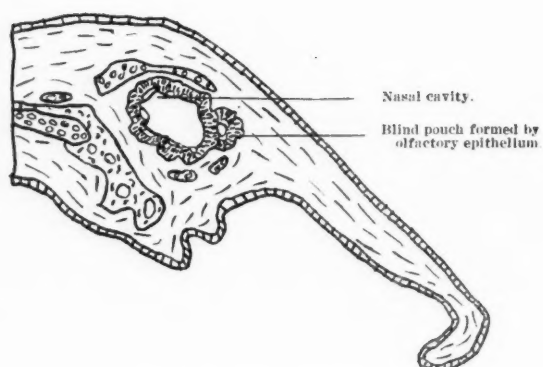


Fig. 3. *Nectures maculatus*.

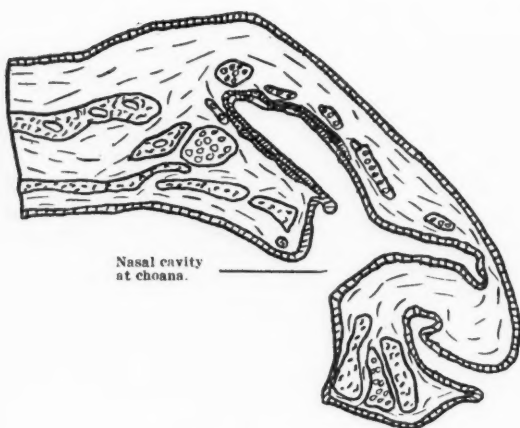


Fig. 4. *Nectures maculatus*.

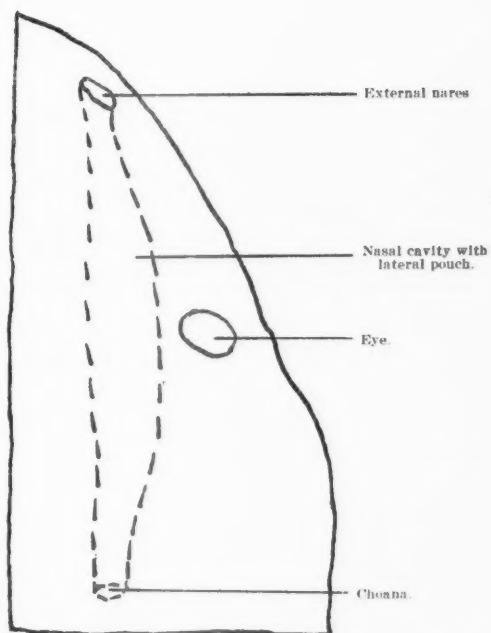


Fig. 5. *Amphiuma tridactylum*. Diagrammatic dorsal view.

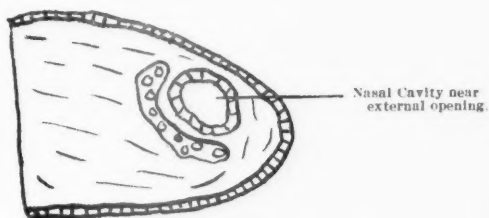


Fig. 6. *Amphiuma tridactylum*.

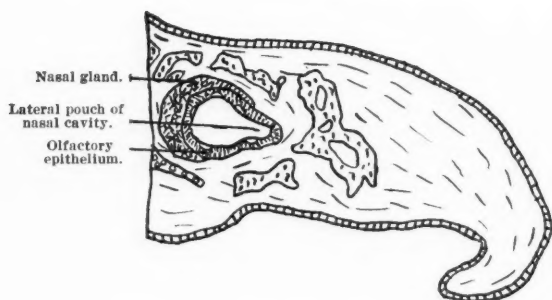


Fig. 7. *Amphiuma tridactylum*.

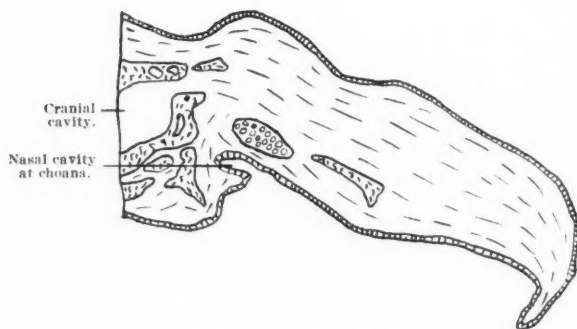


Fig. 8. *Amphiuma tridactylum*.

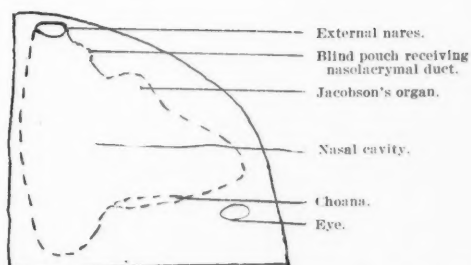


Fig. 9. Salamander (*Plethodon Glutinosus*). Diagrammatic dorsal view.

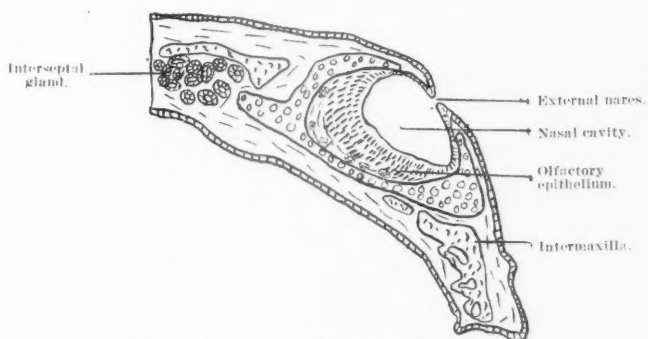


Fig. 10. Salamander (*Plethodon Glutinosus*).

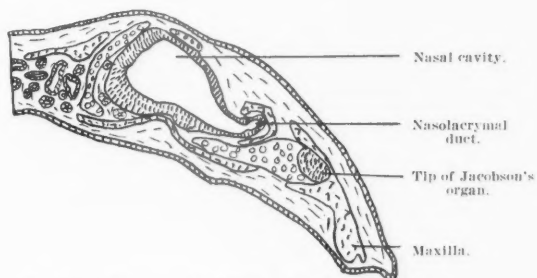


Fig. 11. Salamander (*Plethodon Glutinosus*).

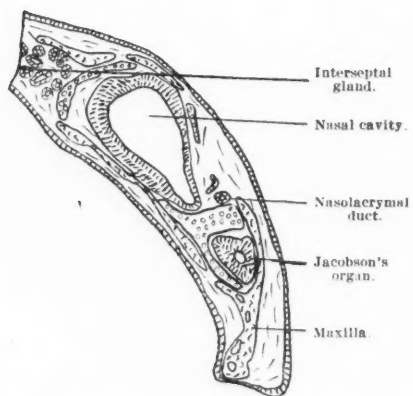


Fig. 12. Salamander (Plethodon Glutinosus)

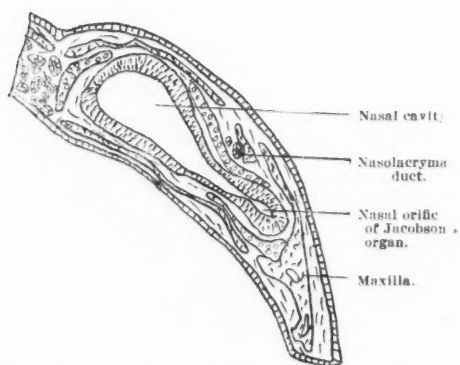


Fig. 13. Salamander (Plethodon Glutinosus)

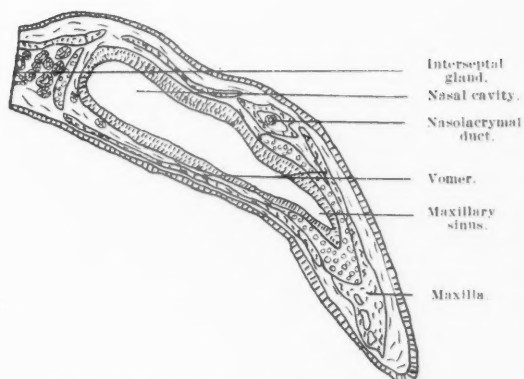


Fig. 14. Salamander (Plethodon Glutinosus).

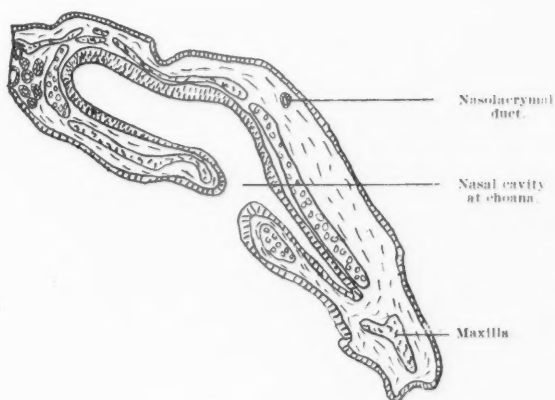


Fig. 15. Salamander (Plethodon Glutinosus).

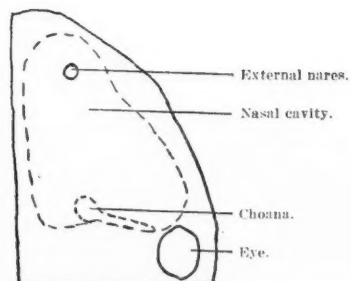


Fig. 16. Frog (*Rana pipiens*). Diagrammatic dorsal view.

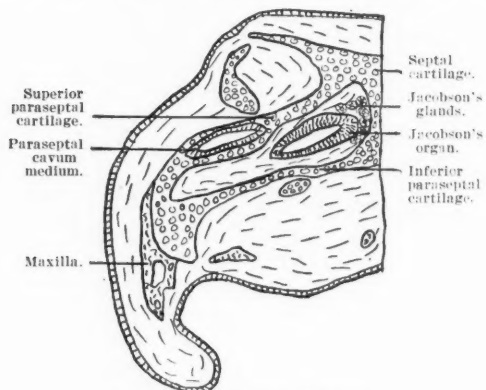


Fig. 17. Frog (*Rana pipiens*).

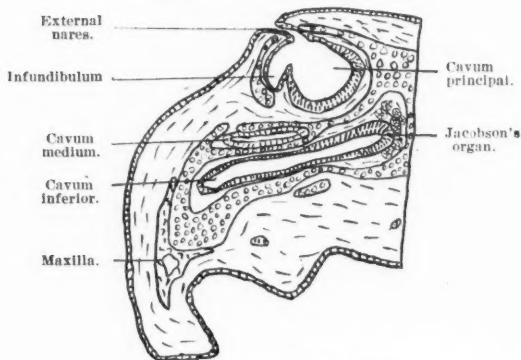


Fig. 18. Frog (*Rana pipiens*).

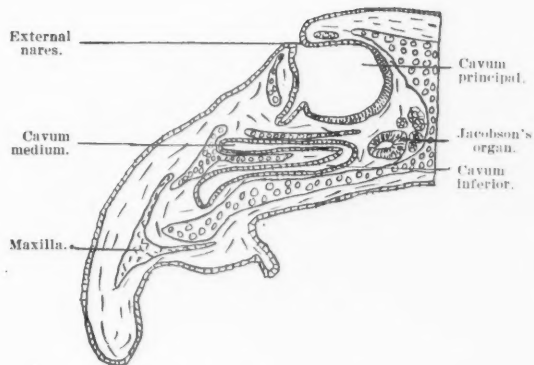


Fig. 19. Frog (*Rana pipiens*).

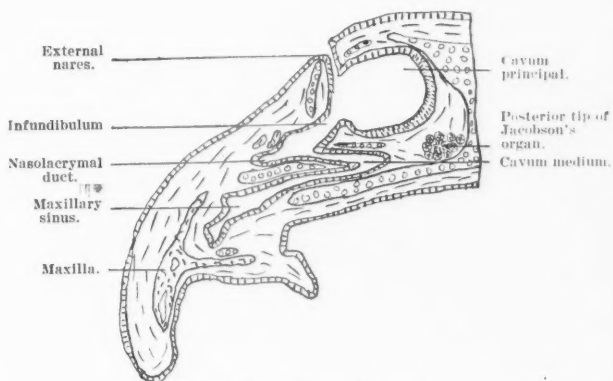


Fig. 20. Frog (*Rana pipiens*).

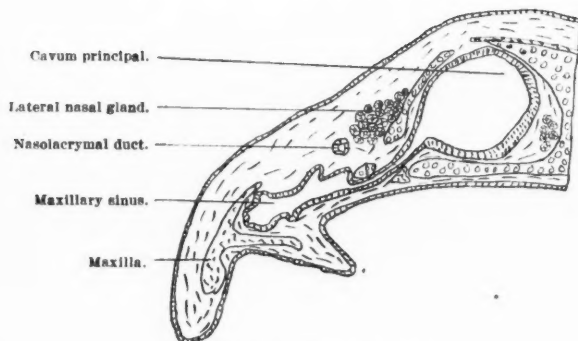


Fig. 21. Frog (*Rana pipiens*).

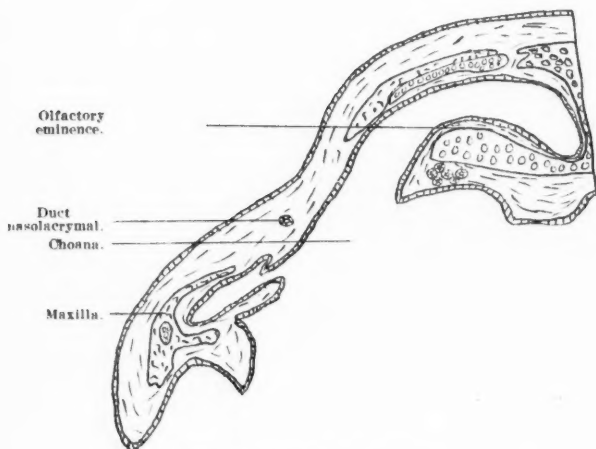


Fig. 22. Frog (*Rana pipiens*).

XL.

TRACHEOTOMY: SOME OBSERVATIONS AND
EXPERIENCES.*

BY MERVIN C. MYERSON, M. D.,†

NEW YORK.

The term tracheotomy was first suggested by Laurence Heister,¹ in 1768.

Up to this time the operation was called laryngotomy and bronchotomy. The first specific description of this operation can be traced back to the early Roman times. We are led to believe, however, that the operation was practiced much earlier than that.

Diphtheria had more to do with the development of the operation than any other condition. The first successful tracheotomy for diphtheria is credited to Bretonneau, in 1825, although Home is credited with having performed this operation in a case of croup in 1765. Prior to this time opening of the larynx or trachea was carried out principally for foreign bodies and for intense angina. The work of Bretonneau was quickly taken up by others, among whom was his pupil Trouseau. The indications for this operation were apparently not very well defined, for in many of the earlier works one sees tracheotomy advocated for cases of drowning.

The development of this operation constitutes a fascinating chapter in the history of medicine.‡

Various methods were devised to overcome dangerous or objectionable features of the operation. For more rapid operation, a trocar and cannula were used by a few. To overcome excessive bleeding, the cauterly or chemical agents were used

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‡Two volumes of reprints on the subject, collected by Dr. Lewis Stephen Pilcher, and presented to the Kings County Medical Society Library of Brooklyn, are unusual for their content and the period which they cover.

to perform the operation. Some used the cautery after the tissues had been cut. To eliminate the possibility of erosion of the innominate artery, soft rubber tubes were advocated to be used as cannulae. One author performed tracheotomy and then approximated the skin and tracheal edges so that a cannula was not necessary.

Tracheotomy is required to overcome obstruction to the passage of air through the larynx or upper trachea. One could list many causes for obstruction in this region. The laryngologist must, of course, be conversant with local changes in these structures and the diseases which may cause such changes. These changes are caused by paralyses, various types of inflammation, and infiltration, foreign body lodgment, wounds and pressure from without.

Tracheotomy is usually a procedure which is urgently required. Occasionally it is performed as a matter of expediency or of choice, preparatory to some other operation. Tracheotomy is sometimes performed as a preliminary to thyrotomy where a large amount of bleeding is anticipated. Or this operation may be advisable as a preliminary to an extensive endolaryngeal procedure by means of the suspension apparatus.

Today, because of the universal recognition of the value of bronchoscopy in foreign body work, tracheotomy is not resorted to for bronchial foreign bodies as it formerly was. It is, however, employed by some in the delivery of movable foreign bodies in the trachea. Some experiences with movable foreign bodies in the trachea have caused the writer to open the trachea in all cases of this kind. It has been found greatly to the advantage of the patient to deliver the intruder through a tracheal incision, rather than to attempt its removal by means of a tracheoscope or bronchoscope. Such foreign bodies as beads, peas, beans, plum stones are more readily removed through an opening in the trachea than by means of peroral endoscopy. This stand is acceptable to many bronchoscopists and does not meet with the approval of others. One must decide for himself what it is best to do under given circumstances, and his decision is dependent upon his judgment, backed by his training, knowledge and experience.

There are two reasons why tracheotomy should be performed when an ovoid or round foreign body is shooting back and

forth in the trachea. First, there is usually such a severe reaction on the part of the subglottic mucous membrane which is being struck by each movement (shot) of the foreign body that tracheotomy is usually necessary even if the intruder is readily extracted from the trachea.

The second reason is that this type of foreign body is very difficult to grasp and usually requires extra manipulation. Such being the case, the trauma of the manipulation is added to the trauma which the foreign body has caused and urgent laryngeal dyspnea ensues. Where the foreign body is a bead, bean or pea, the mere opening of the trachea will permit the patient to cough the intruder out. The tracheal tube is removed within forty-eight to seventy-two hours and recovery is uneventful.

The question of when to perform a tracheotomy is as important as the operation itself, for if too long delayed tracheotomy may be performed in vain. This has been known for centuries, yet we see cases where the operation is withheld until the patient can no longer react favorably to the opening of the trachea. The storing up of an excess of carbon dioxide in the blood and the gradual loss of oxygen from the blood and tissues soon bring the patient to a state where his respiratory center can no longer react.

Haldane² says that if the amount of carbon dioxide in the blood is greatly reduced by artificial respiration there occurs an apnea and the animal dies from want of oxygen, without breathing. He calls attention to the fact that voluntary forced breathing causes apnea; that as a result of the combined stimulus of carbon dioxide and the lack of oxygen, the hemoglobin runs down so low that the lips and face of some persons become alarmingly blue before breathing begins. In speaking of the experiments of Yandell Henderson along these lines, Haldane says: "When the carbon dioxide pressure is reduced below a certain point in the respiratory center the latter ceases to respond even to the extremest stimulus."

Negus³ says: "It is apparently the sudden withdrawal of carbon dioxide from the alveolar air and the consequent inactivity of the respiratory center, temporarily set to respond to a less alkaline blood reaction caused by the higher percentage of carbon dioxide, that leads to danger in tracheotomy."

Here Negus discusses the matter of patients dying as a result of the late opening of the trachea.

On the other hand, there are occasions when it is well to spare the patient a tracheotomy. This is especially true in individuals who are seriously ill with some disorder other than partial obstruction to the airway, or where local conditions of inflammation, especially, make it desirable to avoid surgery in the immediate locality. In Ludwig's angina it is always desirable to avoid tracheotomy if possible. Here we are dealing with a streptococcic infection of the floor of the mouth, and incision into the uninfected deep planes of the neck should be avoided if possible. In such cases, when edema of the laryngeal structures develops and there is interference with the airway, it has been the writer's practice to introduce a silk woven catheter, 20 to 28 French, into the trachea by means of direct laryngoscopy. The catheter is made fast to the skin of the face and is removed at the end of twenty-four to forty-eight hours, when the edema has usually subsided as a result of operation by the surgeon.

On two occasions I have sat at a bedside all night with the hope of avoiding tracheotomy in a child. Tracheotomy was avoided, but it is questionable whether the suffering of the patient and the anxiety of the physician in such cases should not decide in favor of operation.

There are certain conditions in which tracheotomy is sometimes erroneously performed. One should not permit himself to be hastily impressed into performing this operation upon patients who are dyspneic because they are terminating from a cardionephritic condition, a fibroid phthisis, or, in the case of infants, a polioencephalitis involving the respiratory center, or an extensive pneumonia. In all these conditions, especially in a pneumonia which is sudden in onset and which spreads rapidly and is very extensive, there may be an intense dyspnea, with or without cyanosis, which may mislead one into assuming that a laryngeal or upper tracheal obstruction exists. In all patients where the question of tracheotomy is considered, the routine examination with the stethoscope over the open mouth and over the trachea elicits what is probably the most valuable diagnostic sign. Inspiratory stridor is heard below the larynx. Expiratory stridor is heard at the open mouth. The point of

maximum intensity of the inspiratory sound will help localize the approximate point of obstruction. Retraction, real or apparent, may be present because of severe dyspnea or because of obstruction within the chest, not necessarily laryngeal or upper tracheal obstruction. Retraction of the suprasternal, supraclavicular, infrasternal and intercostal areas is always present in severe obstruction to the larynx or upper trachea.

The recent addition of the oxygen tent and the oxygen chamber to our equipment for the treatment of cases of respiratory distress has eliminated the necessity for many tracheotomies. Vinson⁴ has repeatedly stressed the value of this adjunct following the extraction of foreign bodies from the bronchi of infants or young children. Our experience has been the same as that of Vinson. He places all infants and young children who have been bronchoscoped into the oxygen chamber immediately after bronchoscopy.

Moersch and Boothby⁵ are enthusiastic advocates of this therapeutic agent.

The employment of the bronchoscope to pipe air down to the lung of a suffocating patient as a preliminary to tracheotomy has eliminated the necessity for rapid tracheotomy and its disadvantages. Lynah⁶ and Arrowsmith used this form of preliminary intubation several years ago, and the writings of Gill and Lewis have brought this procedure to the attention of the profession. Jackson advocates this procedure.

The use of the bronchoscope as an aid to tracheotomy is not without its dangers. If the bronchoscopist is not sufficiently adept in handling the tube, the manipulation, together with the delay, may cause him to lose his patient. The bronchoscope must be passed rapidly, and the respiratory distress must not be increased by unnecessary or awkward manipulation. I have seen a patient with carcinoma of the larynx expire while repeated attempts were being made to pass a bronchoscope through his larynx. In this case there was sufficient space for the passage of the bronchoscope, but the operator could not find the larynx quickly enough. This, of course, should not occur in the hands of a skilled bronchoscopist. Therefore, bronchoscopy as a preliminary to tracheotomy for urgent dyspnea should be performed only by one who can be certain to pass

the tube. This is one place where repeated attempts must not be permitted.

When the bronchoscope is passed, however, the operation of tracheotomy is greatly simplified, for an emergency procedure has then been transformed into a deliberate one.

For emergency relief as a preliminary to tracheotomy, the indirect cannula or life saver of Mosher and the laryngeal tube of Schrotter are worthy of mention. The Schrotter tube, being made of hard rubber, has the disadvantage of breaking when severely stressed. It is also very difficult to manufacture. It is therefore suggested that metal tubes of the same shape and suitable sizes as the Schrotter tubes would be of advantage.

In Case 4, a Schrotter tube was passed through the severely obstructed larynx of a patient who was transported ten miles with the tube in place to the hospital where tracheotomy was performed. The hard rubber tube broke off at the incisors and, therefore, added to the difficulty of steadying it in place.

The value of operating along the exact midline of the neck was recognized by all the earlier writers. Present day surgery makes the operation of tracheotomy simple and without difficulty or complication. Median and low tracheotomy are practiced universally today. As time goes on high tracheotomy will be mentioned mainly to be condemned, and will be reserved for cases where a stab through the cricothyroid membrane is imperative to save life.

A midline incision with division of the fascia and separation of the pretracheal muscles and exposure of trachea, always staying in the midline, is the key to successful tracheotomy. Either resection of the isthmus of the thyroid gland or pushing it up out of the way can be practiced if this structure happens to be in the path of the operation. One can expose the cricoid area, together with the upper tracheal structures, and make a transverse incision into the pretracheal fascia. This will permit the passage of a pair of Mayo scissors behind the thyroid isthmus, at the same time elevating the pretracheal fascia from the trachea, then double clamping and resection of the isthmus, followed by suture ligature, will take that structure out of the field of operation. The trachea will then be exposed

and incision in the region of the fourth and fifth rings can be carried out. In the operation, as practiced by the writer, the cricoid area is not exposed purposely. In only an exceptional case has it been found necessary to cut through the thyroid isthmus. As a rule, this structure can be readily pushed up out of the way. The excision of a portion of the central area of the tracheal rings has been found of value in more easily and properly placing the tracheal tube. Pieces of cartilage are frequently broken off from the tracheal rings when only an incision is made preliminary to inserting the tube.

The postoperative care of these patients is important. Intelligent nursing is of great value. Tracheotomy patients usually have a reaction that is in direct proportion to the acuteness and the degree of obstruction which comes on rapidly rather than gradually. They invariably develop a tracheobronchitis after operation. This is evidenced by the increased bronchial secretion and its greater viscosity. This condition is sufficient to cause a rise of the temperature to 102 or 103 and an increase in the rate of respiration.

A diagnosis of pneumonia is frequently made, only to be proven erroneous by the return of the temperature to normal or almost so within forty-eight hours.

The use of a suction apparatus and a catheter which will not completely block the tracheal tube is imperative and is as essential as the scalpel is for incising the skin. The frequency of the application of the suction should be dependent upon the quantity of fluid that accumulates in the tracheobronchial tree in a given time. Some cases require the application of suction quite frequently, while others need very little of it. We have been instructing our nurses to listen over the tracheal tube for coarse rales or a delicate gurgling sound that denotes an accumulation in the bronchi. A fixed rule for the frequency of suction insures its use, but also exposes the patient's respiratory tract to unnecessary trauma. The room should be kept at a temperature of 70 degrees Fahrenheit.

Steam vapor is of value in a room that is known to be too dry, or in a case where the bronchial secretions have a tendency to become inspissated. In the average case the employment of steam has no special value. Barling,⁷ who had a considerable experience with tracheotomy cases, expressed the

same sentiment almost fifty years ago. He observed that moist warmth depressed the patient, and that the condensing steam made the bed clothing wet.

The use of a wet sea sponge or a curtain of several layers of gauze over the tracheal tube orifice is of value. The writer has always preferred four or five thicknesses of gauze suspended by a piece of tape or bandage tied about the neck. The gauze can be replaced when soiled, while the sponge must be washed frequently, and is not as easily handled.

Patients who are destined to wear their tubes permanently can be taught to use some form of speech apparatus, such as that devised by Mackenty. One of my patients with an apparently cured carcinoma of the thyroid gland uses such an apparatus to great advantage.

The following cases are cited as interesting and unusual:

Case 1.—S. A., male, white, age 43, had an emergency tracheotomy performed with the aid of a preliminary bronchoscopy by means of the Yankauer-Killian 9 mm. tube. He had been hoarse for three months, he had painful phonation for one and a half months, a loss of weight of thirty pounds during the past three months, and sleeplessness, because of dyspnea, for the past two weeks. He had been operated upon five years before for rectal fissure. During the past year he had used his voice excessively for twelve hours a day as a bus starter. Three months ago he was referred to a hospital, where he received three radium applications, the last application being made to his neck two weeks prior to the time of admission.

Examination revealed that the right eye was the seat of an old keratoiritis. Chest examination showed that there were diminished breath sounds and crackling rales at the right apex. The larynx revealed marked swelling and edema of all the visible structures; the vocal cords could not be seen. The Wassermann reaction was reported as four plus. The sputum was negative for tubercle bacilli. X-ray study showed what was interpreted as disseminated tuberculosis of the lung.

The provisional diagnosis was carcinoma of the larynx.

A specimen removed from the larynx was reported as acute inflammatory tissue. A second specimen removed five days later was reported back tuberculosis.

The patient was discharged after remaining in the hospital thirty-six days with a cannula still in his trachea. At the time of discharge there was considerable thickening of both false and true vocal cords, and the air space was insufficient to permit decannulization. Two months later, however, the tracheal tube was removed without incident. The larynx was functioning perfectly, except for slight thickening and some irregularity of both true cords and the left arytenoid region.

Comment.—This case was originally treated for carcinoma because it was not thoroughly studied. We were not certain of the absence of carcinoma, despite the other findings, until a biopsy was performed. The importance of proper and complete study cannot be overestimated. Complete rest of the larynx from its speech and respiratory function was of real value in overcoming the local reaction in this case.

Case 2.—M. T., female, white, age 21, was admitted to the hospital, where emergency tracheotomy was performed. She had great difficulty with breathing for the past three days, had been hoarse for two months, and was experiencing a burning sensation in the throat for the same period of time. She was in her eighth month of pregnancy.

Her past history revealed that she had been in a tuberculosis sanitarium two years ago for five months.

Tracheotomy was performed after a preliminary bronchoscopy with a 7 mm. Yankauer-Killian bronchoscope.

The chest examination revealed fine rales in both apices, and X-ray of the chest showed infiltration of both upper lobes. The Wassermann reaction was negative. Three days after operation premature delivery of the infant was carried out. Cauterization of the edematous epiglottis and arytenoids was instituted. This was done twice, seven days apart. Two weeks later the patient was decannulated. She was discharged with a well functioning larynx and the edematous reaction had disappeared after six weeks.

Comment: This case demonstrates what is well known, namely, that pregnancy aggravates tuberculosis of the larynx. The beneficial influence of tracheotomy upon tuberculosis of the larynx is demonstrated.

Case 3.—F. P., female, colored, age 36, was seen on the pneumonia service of Dr. Bullowa at the Harlem Hospital.

Her entire right chest was involved in a lobar pneumonia. Nine days after admission, when her pneumonia was apparently subsiding, she developed slight hoarseness. Two days later the chest was clear, but slight hoarseness persisted. During the afternoon of this day she developed marked inspiratory stridor.

Direct laryngoscopy revealed a generalized edema with very little air space. Tracheotomy was performed by Dr. Kleinfeld, after a preliminary bronchoscopy. Three days later examination revealed a patch of white exudate in the region of the vocal process of the left arytenoid. The left arytenoid was swollen two and one-half times its normal size, with edema extending into the aryepiglottic ligament. The right arytenoid was also somewhat edematous. Six days after tracheotomy the edema of the right arytenoid had subsided. The left arytenoid was markedly infiltrated and immobile, and presented a granuloma on its anterior aspect. The granuloma became smaller but the swelling in the left ventricle persisted.

A Kahn test for lues was reported as positive. Antiluetic treatment was instituted. The process in the larynx disappeared rapidly. The patient was decannulated and discharged as cured within a month. At the time of discharge there was some thickening and loss of motion of the left arytenoid.

Case 4.—W. M., male, white, age 34, was sent into the hospital during the night after the writer had placed a Schrotter tube into his larynx. Tracheotomy was performed with this tube in place. The patient was seen at home, four years before, where he had a progressive laryngeal obstruction due to what appeared to be a generalized edema of the larynx. He was hospitalized and scarification of the edematous areas was carried out as an emergency measure. The edema disappeared. Breathing was again unimpaired.

Examination disclosed a slightly distorted right false cord and a thickened right arytenoid. Lues was thought of, but repeated Wassermann tests were negative. A provocative Wassermann test revealed a one plus reaction. Salvarsan was given intravenously, and the patient developed a lung abscess following one of the intravenous administrations. This lung suppuration was cured by bronchoscopy. The patient refused further antiluetic treatment. Two years later he returned with

a large ulcer on either leg. These were definitely luetic and healed after protracted antiluetic therapy. He felt so well that he disregarded the treatment outlined for him. He accordingly came back one day, rather hoarse. Examination revealed the presence of an edema of the right side of the larynx spreading across to the left arytenoid. He was referred for immediate antiluetic treatment. Two days later he was seen early in the morning. He had apparently stopped breathing when the writer forced an indirect Schrotter tube down into his larynx. He was taken to the hospital, where tracheotomy was performed and antiluetic treatment in the form of bismuth given. After the first intramuscular dose the edema disappeared rapidly. Twenty-one days after tracheotomy the tracheal tube was removed. He now has a markedly distorted larynx, both true cords being irregular and the right arytenoid fixed.

Comment on Cases 3 and 4: Luetic involvement of the larynx is uncommon, since the Wassermann reaction and modern antiluetic therapy have come into use. The appearance of increasing edema of the larynx should lead one to suspect lues. When tuberculosis and carcinoma are ruled out it is well to give antiluetic treatment, even in the presence of a negative Wassermann, in some cases.

Case 5.—M. F., male, white, age 12 months, had had a croupy cough for two weeks with increasing dyspnea. On admission there was severe inspiratory dyspnea.

Bronchoscopy revealed a slight exudate on the walls of the trachea and bronchi, and a narrowing of the lumen due to a moderate degree of edema. The opening of the right main bronchus was reduced to about two millimeters, owing to the swollen mucosa. No foreign body was seen, but the mucosa of the entire bronchial tree was markedly reddened. After bronchoscopy dyspnea and cyanosis were markedly increased. This necessitated tracheotomy, which gave apparent relief. At the time of tracheotomy, a few hours after the bronchoscopy, subglottic edema was noted. The child was placed in an oxygen tent but expired fourteen hours later.

Autopsy revealed diffuse bilateral mucofibrinous bronchitis with atelectasis of both upper lobes and part of the remaining

lobes. Bacteriologic examination revealed a short chain streptococcus.

Comment: This was a case of streptococcic tracheobronchitis in which sudden and intense dyspnea misled us into suspecting the presence of a foreign body.

Case 6.—C. R., female, white, age 65, had been suffering with diabetes for the past three years. She was admitted to the hospital because of acute respiratory distress, and presented marked dyspnea with some cyanosis. The laryngeal examination showed that both cords were in the midline. There was an inability to abduct. Diagnosis: Bilateral abductor paralysis.

Two weeks ago she first became hoarse and later became dyspneic. The hoarseness lasted two days. She had a good speaking voice when first seen at the hospital.

The Wassermann reaction was found to be four plus.

Tracheotomy was performed after a preliminary bronchoscopy. The acute distress was immediately relieved. Treatment was instituted for the lues and diabetes. The local laryngeal picture cleared up, and decannulization was possible twenty days after tracheotomy. The patient was discharged with an apparently normal larynx.

Comment: The restitution to normal of a larynx which was previously involved in a bilateral abductor paralysis has been reported but is very uncommon.

Case 7.—R. E., female, white, age 50, was in good health until nine months ago, when she noticed that her neck was stiff, and that her face was swollen and she was unable to rotate her head. During the past three months she had suffered severe attacks of dyspnea. She also complained of a non-productive, spasmodic cough. At the beginning of her illness an X-ray examination revealed a mediastinal shadow. X-ray therapy caused her symptoms to disappear, but three months ago they returned. A mass appeared on the right side of the neck, and cough and slight expectoration were present. At this time she complained that food appeared to stick at the level of the upper end of the sternum (upper thoracic opening). At this time stridor was present during inspiration, and attacks of dyspnea were frequent.

Examination of the larynx on the day after admission showed the presence of a right recurrent laryngeal paralysis. Twenty days later the other side of the larynx was involved in a similar manner so that the patient now presented a bilateral recurrent laryngeal paralysis. Bronchoscopy disclosed a trachea turned somewhat to the left. At a point twenty centimeters from the upper teeth the left tracheal wall showed an irregular infiltrative process, submucously placed, apparently extending from without. No bronchial compression or endobronchial disease was found.

Tracheotomy was performed with the bronchoscope in place. The trachea was found twisted so that the right lobe of the thyroid gland occupied a middle position. It was found necessary to resect a portion of this structure.

On the sixth day after tracheotomy there was sudden and copious bleeding from the tracheal wound and the patient ceased to breathe. A large vessel had evidently become eroded by the malignant infiltration.

Autopsy was not obtainable. It was thought that the patient probably had a sarcoma of the mediastinum with involvement of a right subclavian gland to account for the bilateral recurrent nerve involvement.

Comment: A bilateral recurrent laryngeal nerve paralysis is extremely rare.

Case 8.—R. M., female, white, age 4 months. Eight weeks ago the baby became hoarse. Four weeks ago the glands of both sides of the neck became swollen, and at this time respiration became noisy.

On admission a large swelling was palpable in the pharynx on the right side. This proved to be an abscess, which was opened, and about two drams of pus were evacuated. Three days after admission the left upper chest, posteriorly, was dull to percussion. At this time the hemoglobin was 85 per cent, and there were 19,600 white blood cells with 78 per cent of polymorphonuclears. The spleen was palpable. A few days later a serosanguinous discharge was seen to be coming from the right ear, and the left ear drum was found to be full. The child became intensely dyspneic on the seventeenth day. Tra-

cheotomy was performed by Dr. Kleinfeld, but the child died a few hours later.

Autopsy revealed a right retropharyngeal abscess cavity empty and almost healed; purulent exudate in both ethmoid regions; purulent bronchitis with bronchopneumonia of all lobes. Pus from the ethmoid regions and bronchi showed *streptococcus viridans* on culture.

Comment: Tracheotomy was performed in this case because of the intense dyspnea which the child developed. This dyspnea was due to an involvement of all the lobes of the lung in a bronchopneumonia. The coincidence of ethmoidal and bronchial suppuration is noted. Tracheotomy was not indicated.

Case 9.—S. G., male, white, age 43, became hoarse four days before admission and at the same time he experienced some difficulty in swallowing. He was now quite dyspneic and presented an inspiratory stridor.

Direct laryngoscopy revealed a large acutely edematous swelling of the left arytenoid region and aryepiglottic ligament, extending downward into the larynx and overhanging the glottis. This edematous area was punctured and a few drops of pus were evacuated. Forty-eight hours later the patient again developed urgent dyspnea, and tracheotomy was decided upon. Bronchoscopy was performed preliminary to tracheotomy. During the tracheotomy a small amount of pus was evacuated in the left lateral cricothyroid region. The patient remained in the hospital about two weeks and was sent home with the tracheal tube in place. Two months later decannulization was carried out. The left side of the larynx was found to be fixed, the left arytenoid slightly thickened, and the left aryepiglottic ligament slightly shortened. One year later the same picture is present, with perhaps a little less thickening of the involved structures.

Comment: The abscess of the larynx in this case was very likely due to a bone or other sharp foreign body which carried infection with it. No foreign body was seen at any time.

Final comment: Several phases of the operation of tracheotomy are considered and nine interesting cases are cited.

12 EAST 86TH STREET.

BIBLIOGRAPHY.

1. Heister, Laurence: *Bronchotomy, Laryngotomy or Tracheotomy. A General System of Surgery*, 8th Edition: London, 1768, Vol. II, Part 2, Sec. 3, p. 51, ch. 102.
2. Haldane, J. S.: *Respiration*. New Haven: Yale University Press, 1922, p. 111. See also Chapter 8.
3. Negus, V. E.: *Some Disorders of the Larynx*. Hunterian Lecture. *The Lancet*, Sept. 19, 1925, pp. 581-86, CCIX.
4. Vinson, P. P.: Personal communication.
5. Moersch, H. J., and Boothby, W. M.: *The Value of Oxygen Following Bronchoscopy in Children*. *Arch. Otolaryng.*, December, 1927, Vol. 6, pp. 542-545.
6. Lynah, Henry L.: *A Series of Foreign Bodies in the Bronchi and Esophagus*. *Trans. Am. L., R. and O. Soc.*, 1919.
7. Barling, Gilbert: *Notes of Tracheotomy*. *Birmingham Medical Review*, October, 1884, pp. 150-158.

XLI.

A PLEA FOR MORE CONSERVATISM IN THE
TREATMENT OF NASAL ACCESSORY
SINUS INFECTIONS.

BY WILLIAM WESLEY CARTER, A. M., M. D.,†

NEW YORK.

In no department of surgery has more ingenuity in the planning of operations been displayed than in that of nasal accessory sinus surgery, and in no field have physiologic considerations been more often ignored or subsidized to mechanical devices that have destroyed tissues that could not be replaced and effected undesirable anatomic changes that could not be effaced. Besides this, the dangers attendant upon radical sinus surgery are constant and unavoidable, and I feel that they are not sufficiently stressed by writers upon this subject. One should never lose sight of the fact that these operations are major procedures and that when the frontal, the ethmoids or the sphenoid, especially, are involved, the danger of meningitis is a menace that must be recognized by every surgeon doing this work, however, skillful and experienced he may be. We all know this, and we also know that published statistics fall far short of giving a correct estimate of the seriousness of this hazard.

Some of the operations devised for the relief or cure of pansinusitis are marvels of ingenuity and are based upon accurate knowledge of the anatomy of the parts and a true conception of the drainage problems. Some of the results of these procedures, however, show that enthusiasm over the efforts to secure mechanical perfection has caused the operator to destroy much healthy tissue that might have been saved, tissue that had its definite function to perform, a function that could never be fulfilled by the scar tissue which follows its removal.

*Read before the New York Medical Union, March 25th, 1930.

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I have been interested in sinus surgery for many years, and during this time, having had at my disposal excellent clinical facilities, I have performed practically every operation advocated by those whose operative acumen was recognized and whose clinical experience entitled them to speak with as much authority as a medical or surgical subject may be discussed, with true regard for clinical facts, and we know that this is limited, for all of our work, whether it be a medical or surgical, is passed upon by Nature, that inflexible arbiter of all of our efforts.

In all operative procedures the dramatic, artistic and even the mechanical appeal must be subsidized to the good of the patient. If relief or cure can be secured by means of a procedure possessing mechanical and artistic merit, then so much the better, but clinical results, regardless of the method, must always be considered of paramount importance.

Now let us approach this subject in a practical manner, with an open mind and one not to be affected by either professional or scientific pride or any other consideration that does not bear directly upon clinical results. What are the nasal accessory sinuses, anyway? In answering this I am going to disregard anatomic minutiae and the esthetic embellishments with which they are usually invested by those of us who specialize in this field and say that they are essentially air pockets, connecting with the nose by means of tortuous ducts, or else ostia, as in the sphenoid and maxillary sinuses, and many of the ethmoid cells, so placed that the laws of both hydrostatics and gravity oppose natural drainage. We record the truth when we admit that it is chiefly as a menace that they merit our attention. But "thereby hangs a tale," for when these sinuses become infected and their mucous membranes so congested that they become closed cavities, as in acute conditions, or permanent foci of infection when chronicity is established, then our responsibility begins and our ingenuity in giving relief is called into play.

Operative measures in acute sinusitis are not to be considered, the danger of meningitis and pyemia is great, and besides, owing to tumefaction of the tissues, even minor attempts at drainage would not bring relief. The urgency of the symptoms

in an acute exacerbation of a chronic condition may call for immediate surgical drainage, but no more radical procedure is permissible at this time.

Now when it comes to the treatment of old chronic inflammations of the sinuses by radical operative measures, I have some sad admissions to make, and having seen some of the results secured by my most celebrated confreres, I feel that my song of lamentation could easily become a mighty chorus. Many, very many radical operations, by whatever name they may be designated, upon the frontals, the ethmoids and sphenoid and even the maxillary sinuses, done by men of distinction, adequate clinical experience and consummate manual dexterity, leave scabbing, granulating areas, glistening and dry or bathed in ever present foul smelling purulent debris that constitutes a menace to the health and happiness of the patient as long as he lives. We speak with careless joy of having entered the ethmoid body and having obliterated the frontal sinuses and of having successfully converted the maxillary antrum into an alcove of the nasal cavity. These are all mechanical achievements accomplished by skillful hands. But what of the aftermath? We may or may not be able to follow in its wake in every instance and note the final results, but if we have examined some of these patients and noted their postoperative history, our conclusions surmount the hazards of a guess that very few are cured by extensive operations upon the sinuses.

Bare bone does not procure for itself another periosteum, nor does it remain alive. Ciliated epithelium, once destroyed, is never regenerated. Normal mucus glands do not reappear in the scar tissue which follows the destruction of mucous membrane, nor is this scar tissue capable of performing any physiologic function.

The sequence of events in sinusitis are the same as in those of inflammation in any other part of the body. First, there is congestion, which if it continues long enough, is followed by hyperplasia and hypertrophy after the inflammatory process has become chronic. Later this stage of hypertrophy is followed by atrophy and degeneration. Sinuses that have been subjected to repeated attacks of inflammation will show under the X-ray a thickened lining mucous membrane. This is best

brought out by first washing out the sinus and then injecting it with lipiodol before making the exposure. In these chronic cases polypi will frequently be found in the antrum and especially the ethmoid area. But even if polypoid degeneration has taken place, it is no indication that the mucous membrane should be ruthlessly curetted away and the bony walls of an infected cavity left bare. Under such circumstances what chance has Nature to effect a cure? We may conservatively say *none*. We have no more right to scrape away the thickened mucous membrane of the sinuses than we have to destroy the mucous membrane of the nasal cavity. The thickened membranes which we are now discussing are due, not to pathologic conditions originating within these membranes, but to the stimulation of an irritant, usually an infection in the cavity which they line. Therefore, the logical, and I may say the practical cure for sinusitis is the elimination of the irritant, which, in the majority of instances, may be accomplished by conservative operative measures, if necessary, for the purpose of promoting drainage, by proper local treatment and by strengthening the vital resistance by medication and hygienic measures.

Polypi must be removed, for not only do they interfere with drainage, but this tissue has already passed beyond the realm of redemption. The simple removal of these with the snare, however, is not curative, for we all know that they recur with remarkable celerity, especially in the ethmoid region. They are associated with disease of the underlying bone which may be found bare and friable. A discussion of the pathology of polypi does not come within the scope of this paper; suffice it to say, however, that fibrotic changes in the mucoperiosteum, attended by arteriosclerosis, is always present, together with a rarefying and occasionally a condensing osteitis of the cell walls. The foul odor present in most of these cases speaks eloquently of dead bone. Where there is long continued suppuration attended by insufficient drainage, polypi are nearly always present, especially if the patient is past middle age. These furnish *prima facie* evidence that the bony walls of the cells are extensively involved and devitalized bone is certain to be found. Polypoid degeneration, however, is an independent condition: the presence of polypi, by their

irritation and interference with drainage, invites infection and favors its progress. The true polyp is not the result of infection in the sinuses; it is due to pathologic changes in the deeper layers of the periosteum which are primary in their origin.

From what I have said, it is clear that permanent relief cannot be expected without the removal of the dead bone and hopelessly diseased tissue. The removal of this, however, does not conflict with my view, that destructive operative procedures should not extend beyond the limits of the diseased area. This limit, it is unnecessary for me to say, is not always easy to determine, but it is better to fall short of removing all of the dead bone than to go too far and destroy mucous membrane capable of recovery after the underlying, dead bony cell wall has sloughed away or been absorbed. This recovery of mucous membrane, under prolonged local treatment, I have noted on many occasions.

There are some cases of sinus disease where the choice between two evils lies in favor of the radical operation, but these should be most carefully culled from those that may be relieved by less severe methods, such as conservative operative measures for the improvement of drainage, suction, local treatment, improvement in hygienic surroundings and careful attention to the general health, with the object of increasing the resistance to the infection. It is well known that climatic changes will sometimes clear up a severe sinusitis very quickly.

In many cases of long standing sinusitis, attended by polyp formation, I have found the following conservative treatment satisfactory, and I believe that it, or some similar measure, should be given a thorough trial before resorting to the radical operation.

1. Correct the deflected septum or any other condition interfering with good drainage.
2. Remove all polypi and uncap the affected ethmoid cells.
3. If the antrum is serving as a reservoir for pus, make a large, permanent opening into the inferior fossa for drainage.
4. Empty the sinuses by suction twice a week at least and apply a 20 per cent solution of argyrol.
5. Have the patient cleanse the nose each day with physiologic solution of sodium chloride, and distill into each nostril five drops of 10 per cent argyrol solution.

6. Necessary attention should be given to the general health of the patient. This may, in some instances, include climatic changes.

Usually this treatment must be carried out for several months, but my experience has been that the results are far better than those following radical operations, for, even if a complete cure is not effected, there has been no unnecessary destruction of normal tissue, the patient has been made comfortable, and the nasal cavities have not been converted into voluminous caverns, the walls of which are continuously covered with pus or scabs.

I have never seen a case of argyriasis following the long continued use of argyrol in the nose, and I believe that this occurrence must be extremely rare.

I trust that this cursory review of so important a subject may be considered justified by my earnest desire to impress upon my confreres what I sincerely believe to be a fact: that radical surgery upon the sinuses is rarely followed by complete cure, my conclusion having been reached from observation of my own clinical results as well as those of other operators. I believe, therefore, that rational conservatism should be more rigidly applied when dealing with infections of the sinuses.

2 EAST 54TH STREET.

XLII.

THE UPPER RESPIRATORY TRACT AS A GUIDE
TO NUTRITIONAL DISASTERS.

BY D. C. JARVIS, M. D.,

BARRE, VT.

I might say, by way of introduction, that several papers might have been prepared on this subject in earlier years save for one thing.

A number of years ago, while attending the Trudeau School of Tuberculosis held annually at Saranac Lake, New York, I was assigned as roommate the vital statistician of the state of New York. Later, until his untimely death from influenza, he was vital statistician for the League of Nations. During the six weeks we roomed together he taught me many things.

I told him that as physician, having local charge of the work of the Committee on Mortality from Tuberculosis in the Dusty Trades, representing the National Tuberculosis Association, I had just completed an examination of the upper respiratory tracts of 500 men and had inquired their food selection representing the three meals of the day. I had also examined the upper respiratory tracts of 60 babies representing the offspring of some of these men, and suspected from the work done that a relationship existed between the food selection of the individual and the appearance of the upper respiratory tract. During the six weeks we were together we talked over these observations a number of times. At our parting he made a number of requests.

The first request was that on my return home I examine the upper respiratory tract and inquire into the daily food selection of individuals representing the various decades of life. Having made these observations, he wished me to correlate the observations made on the various ten-year groups one with the other.

The second request was to study in like manner groups living in a city environment, groups living in the environment

of the country and those living in a public institution and to correlate the observations.

The third request was the following: If food selection influenced the appearance of the upper respiratory tract, then it likewise probably influenced the well being of the individual. I was to endeavor to discover whether a syndrome existed and whether a relation existed between the food selection, the syndrome and the appearance of the upper respiratory tract.

The fourth request included the following: If my observations, deductions and conclusions had been correctly made and the various problems in multiple correlation correctly done, then I ought to be able, after an examination of the respiratory tract, to outline to the individual his food selection and the state of his well being. A change in food selection ought to bring about a change in the appearance of the upper respiratory tract and cause a change in the state of the well being of the individual.

As a parting request, he asked me to wait from two to four years after the completion of the work in order that time might test my conclusions.

It has required fourteen years to carry out these requests. I bring only that which has stood the test of time. In carrying out the first request, in addition to studying office and hospital patients, I assumed the responsibility of the care of the students of a private school for three years. These students ranged in age from fourteen to eighteen and were prepared by the school for college.

In order that I might have contact with an older group, I assumed the medical care of students in a private school, having a teacher's training course. This care was for a two-year period.

In order that I might have contact with a more rugged type of individual, I have assumed for five years the medical care of a university football squad, traveling with this squad on its out of town games in order that I might have a close contact with them in a medical way.

The above groups, plus individuals studied in hospital and office, gave the different ten year periods requested.

The observations made are as follows: In the group from ten to twenty, lymphoid tissue seemed to be present in proportion as fats were absent from the diet.

In the group from twenty to thirty, there seemed to be a catarrhal discharge in varying amounts, which seemed to be dependent upon a lack of proper amount of leafy vegetables in the food selection.

In the group from thirty to forty and upwards, there seemed to be a granular condition of the posterior pharyngeal wall dependent upon an excess of cereals and foods made from flour.

In doing the problem in multiple correlation between these groups, as requested by my friend, I found there was little I could hold fast to. While absence of fats seemed to be associated with increase of lymphoid tissue in the ten to twenty year period, no such relationship held in the groups beyond thirty.

While a catarrhal discharge in varying amounts seemed dependent upon a lack of leafy vegetables in the twenty to thirty group, no such relationship held in the other groups.

While a granular condition of the posterior pharyngeal wall seemed in the thirty to forty group to be dependent on an excess of cereals and foods made from flour, no such relationship held in other groups.

The only fact remaining after doing my problem in multiple correlation was the following: No matter how much the upper respiratory tract varied in appearance in the majority of individuals without other evidence of clinical disease, there was a marked increase in redness of the mucous membrane covering the cartilaginous portion of the nasal septum, and to a lesser degree that covering the anterior pillars and aryepiglottic folds when the food selected was of a certain type.

With this bit of evidence remaining, I did the work all over again, this time studying the upper respiratory tract, using as a food classification acid and alkaline producing foods.

Continued observation disclosed the fact that in all groups when acid producing food represented the greater part of the daily food intake, there was an increase in the redness of the

mucous membrane covering the cartilaginous portion of the septum and often to a lesser degree an increase in redness of that covering the anterior pillars and aryepiglottic folds. In individuals selecting most of their food from alkaline producing food, there was an absence of this marked increase in redness in the locations mentioned above.

As a matter of record, the degree of redness has been expressed in degrees of 1, 2 and 3, using the plus or minus sign before each number; plus 3 indicates a fiery redness. Likewise, minus 3 indicates a corresponding pallor. A, B and C have been used as designating the locations, A referring to the mucous membrane covering the cartilaginous portion of the nasal septum, B to the anterior pillars and C to the aryepiglottic folds. Thus, my friend's request that I correlate my observations brought a different result than otherwise would have been possible.

Let us consider the second request that I study individuals living in the environment of the city, others living in the country and those living in public institutions. Because of my location in a state, 62 per cent rural in population, it was possible to compare the city and the country.

In order that I might study individuals in a public institution, I made weekly visits to the Washington County Tuberculosis Hospital and the Vermont State Hospital for the Insane for a period of six months. In correlating the observations made the same conclusions as mentioned under the first request held good. City dwellers showed the best upper respiratory tract. I discovered that fruits, vegetables and milk represented an income on the farm and were to be sold to city dwellers. Apples were to be made into cider or sold. On a few farms having a herd of cows condensed milk was being used, all the real milk being sold to the creamery.

With reference to the third request, that if food selection influenced the appearance of the upper respiratory tract, it likewise probably influenced the well being of the individual and a syndrome existed. In order that this request might be complied with, time was taken to inquire the state of well being of individuals. With the passing of time it seemed possible to work out a syndrome, which was associated with

the selection of acid producing food as representing the greater part of the food intake. When this syndrome was present there was a marked increase in redness of the mucous membrane covering the cartilaginous portion of the nasal septum.

First of all, these individuals were tired all the time. The day's work, instead of being a joy, was something to be gone through with. They were irritable and, without wishing to be so, were hard to live with. While they participated in the three meals of the day, there was no zest for food. They were subject to indefinite symptoms in various parts of the body which were of short duration. When they ought to be most awake, like the middle of the day, they were drowsy. When bedtime arrived and they should be sleepy, they were wide awake and at their best. Their night's sleep brought them only a moderate amount of refreshment, coming, as a rule, to the day's work a bit tired. Their skin was dry and their hair came out more than they wished. They were subject to constipation and all seemed to have a feeling that a long vacation ought to be taken so as to feel right again.

* Patients from the rural section remarked that when such a syndrome appeared in a farm horse he was turned out to pasture, that he might return to his original diet, which was the vegetation of the fields. Apparently the above syndrome and the associated appearance of the upper respiratory tract indicates a return on the part of man to his basic diet which consists to a great degree of foods ripened by the sun.

Let us consider the fourth request, which was the following: If my observations, deductions and conclusions had been correctly made and the various problems in multiple correlation correctly done, then I ought to be able, after an examination of the upper respiratory tract, to outline to the individual his food selection and the state of his well being. This it seems possible to do. Given an increased redness of the mucous membrane in the locations mentioned, it is possible to outline to patients the syndrome described as representing the state of their well being. Or, given the syndrome, it seems possible to outline the appearance of the upper respiratory tract. If then the appearance of the mucous membrane is a guide to

nutritional disaster, then a change of food selection ought to change the appearance of the mucous membrane over the cartilaginous portion of the nasal septum and cause the syndrome to disappear. This it does. The time required is from three to six months, more generally six months rather than three. Experience has proven that it is best, when possible, to request patients to report at the office once a week for one month, then once a month for six months, in order to instruct patients and to observe whether the measures instituted are sufficient to produce the desired change in the appearance of the mucous membrane of the upper respiratory tract and to influence the nutritional disaster syndrome. At the end of this time they are requested to report for a check-up if a return of the syndrome is noted.

As for the attitude to be assumed towards these patients: If the mucous membrane covering the cartilaginous portion of the nasal septum is of a fiery redness, noted as plus 3 on the patient's record, then alkaline treatment, the use of citrous fruits and alkaline producing foods are all three used. If plus 2 represents the degree of redness of the nasal mucous membrane then the juices of citrous fruits and alkaline producing food are used. If plus 1 represents the condition, then alkaline producing food only is used.

The usual method where patients live at a distance is to give alkaline treatment, a teaspoonful of sodium bicarbonate in a glass of warm water at 10 a. m., 3 p. m. and 8 p. m. This is continued for three days. At the end of this three day period a glass of lemonade made without sugar from a half a lemon is substituted for the sodium bicarbonate, the lemonade being continued for three days. Sodium bicarbonate is given for one day, then lemonade again for three days, this three and one rotation being kept up for one month. In addition, a printed list of alkaline producing and acid producing foods is given with instruction to select the daily food from the list of alkaline producing food rather than from the list of acid producing food. At the end of one month the patient is requested to report at the office, if possible, for a study of the upper respiratory tract. If this is not possible, then a report by mail as to whether the syndrome is disappearing is requested.

I wish to report four cases as representing types of patients.

Case 1.—A dentist, practicing in a college town, came to the office, stating that his life was a series of physical depressions, being able to practice his profession about three months out of every four. He had tried various medical paths, hoping to gain the ability to practice his profession without these enforced vacations. Feeling some undiscovered focus of infection existed in his upper respiratory tract, he came to the office with the request that I go over his upper respiratory tract. At the end of the examination, which included roentgenograms of his teeth, and sinuses, I told him that I was unable to gather evidence to substantiate his feeling that a focus of infection existed, but the fiery redness of the mucous membrane covering the cartilaginous portion of the nasal septum suggested that he was in the midst of a nutritional disaster and that he selected his food more from acid producing food than from alkaline producing food. He became interested and returned home with a printed food list. Fourteen months later he came to the office to tell me that for one year he had been free from his former physical let downs and had been able to work at his office each day that he wished to. I asked him if I might look at his nose. The fiery redness had disappeared. He was living, I discovered, almost wholly on alkaline producing foods, and in addition to his meals was eating from four to six oranges each day.

Case 2.—A colleague doing the laboratory work at the Barre City Hospital came to the office stating that a constant pain in the small of the back was making riding in his car almost impossible. He had checked himself up at the hospital laboratory, but was unable to find anything wrong. He wished me to go over his upper respiratory tract, fearing lest a focus of infection existed in his sinuses or tonsils. I was unable to find the focus he suspected, but the crimson redness of the mucous membrane covering the cartilaginous portion of the nasal septum suggested he was in the midst of a nutritional disaster. I told him if I was right I ought to be able to tell him how he felt. On describing the nutritional disaster syndrome to him, he replied it represented just how he felt. A change in food selection plus alkaline treatment brought partial relief within two weeks and complete relief in one month. Eighteen months

have passed without a return of the pain in the back. His nutritional disaster syndrome has disappeared.

Case 3.—A physician in an adjoining city referred his wife to the office three years ago. Over the phone he stated that because of lack of nervous energy she had been obliged to give up all social activities. He was unable to find anything radically wrong with her and requested that I go over her upper respiratory tract with a view to discovering a focus of infection. A check-up, including roentgenograms of the teeth and sinuses, failed to discover the focus of infection he sought. I reported to him that the crimson redness of the mucous membrane covering the cartilaginous portion of the nasal septum and the presence of the nutritional disaster syndrome led me to believe his wife was in the midst of a nutritional disaster. I suggested a different food selection, the juices of citrus fruits between meals, a teaspoonful of cod liver oil once a day and alkaline treatment if his daily observation of her case warranted this addition. At the end of six months she was once more assuming her church and club work. This fall her husband called at the office to tell me his wife was a "well woman." He stated that she had studied her food selection closely and had discovered that in order to keep free from the nutritional disaster syndrome she found it necessary to select 75 per cent of her food from alkaline producing foods. If she fell very much below the 75 per cent, her nutritional disaster syndrome began to make its appearance in from one to two months.

Case 4.—The wife of a granite quarry owner in an adjoining town came to the office and requested that I examine her upper respiratory tract for a focus of infection. She stated as her history was gathered that she had been receiving "iron injections" and various forms of treatments, hoping she could regain the nervous energy necessary to care for her home and family. Her upper respiratory tract was found to be negative, as far as a focus of infection was concerned. But the crimson redness of the mucous membrane covering the cartilaginous portion of the nasal septum was present, as was also the nutritional disaster syndrome. It was suggested that she confine her food for one month to alkaline producing food, following the printed food list given her, and that she take an even teaspoon-

ful of sodium bicarbonate in a glass of warm water at 10 a. m., 3 p. m. and 8 p. m., this to be continued for three days. At the end of this time she was to change to lemonade, made from half a lemon, to be taken the same hours as the sodium bicarbonate. For one month she was to continue three days of lemonade and one day of sodium bicarbonate rotation. In addition she was to take one teaspoonful of cod liver oil once a day. Four months from her first visit she came to the office to tell me that for the first time in years she had been able to prepare the Thanksgiving dinner for her family, sit down with them and partake of the food and visit relatives later in the day. Examination of her nose showed a disappearance of the crimson redness of the mucous membrane covering the cartilaginous portion of the nasal septum. She no longer had her nutritional disaster syndrome.

QUARRY BANK BLDG.

XLIII.

NOVOCAIN APPLIED TOPICALLY AS A LOCAL
ANESTHETIC IN NASAL SURGERY: A
PRELIMINARY REPORT.

BY EUGENE NEFF, M. D., AND WALDO B. DIMOND, M. D.,

MADISON, WIS.

Nowhere in the literature are we able to find reference to novocain applied topically as a local anesthetic in nasal surgery. In the June (1915) issue of the *Journal of the Iowa State Medical Society*, Dr. E. P. Weih has an article entitled "Novocain as Used in the Simpler Nose and Throat Operations." Dr. Weih, however, injected the novocain in 2 per cent solution with adrenalin 1:1,000, one drop to each cubic centimeter added.

Because we have had such splendid results with novocain applied topically in nasal surgery and have been able to practically eliminate cocain crystals in our work, we feel that this article is justified.

The novocain which we have used was prepared by the H. A. Metz Laboratories of New York and is a brand of procain hydrochloride, the chemical name being para amino benzoyldiethyl aminoethanol hydrochloride. As prepared by the Metz laboratories, the chemical comes in rather large, coarse crystals which tend to excoriate the delicate mucous membrane of the nose, but this undesirable effect can be remedied by grinding the crystals with a mortar and pestle to a powdery consistency, which makes the substance more soluble but no more irritating or excoriating than the commonly used cocain flakes.

To date we have used this novocain powder with adrenalin 1:1,000 in twenty-seven submucous resections, nine antra meatal operations, seven ethmoidal eviscerations with nasal polypi, three middle turbinectomies, one intranasal frontal operation, and in more than two thousand antrum punctures

with perfect anesthesia. In no instance did we notice any giddiness, hilarity, clamminess, increased pulse rate, death or any other untoward symptoms which are so common when using cocain flakes.

The method we employ in all nasal surgery, with the exception of antrum punctures, consists in first applying a cotton-tipped applicator dipped in 1:1,000 adrenalin and then in the novocain powder to the sphenopalatine ganglion. If this is properly done perfect anesthesia results for submucous resection, ethmoidal evisceration, turbinectomy, and antra meatal operation. In addition to this, however, we apply the powder to the entire septum in submucous operation, to the anterior one-half of the middle turbinate in ethmoid evisceration and turbinectomy, and to and under the inferior turbinate in antra meatal operation. In diagnostic antrum puncture a cotton-tipped probe saturated with adrenalin and dipped in the novocain powder is simply placed beneath the inferior turbinate.

As to the pharmacologic and physiologic action of this particular drug on the mucous membrane of the nose we have as yet no information, but we have imparted our results to these departments of the University of Wisconsin, and they are at present working on this problem. In our next report we hope to incorporate these findings and results.

XLIV.

THE IMPORTANCE OF THE HISTORY IN THE DIAGNOSIS OF BRAIN ABSCESS.*

By FREDERICK T. HILL, M. D.,

WATERTVILLE, ME.

The history is perhaps the one most important factor in any diagnosis. This is especially so in brain abscess, where, in the early stages, so much depends upon subjective findings. And, as in every type of case, this is probably the most neglected portion of the examination. The reasons for this are generally an unwillingness to spend the necessary time or lack of persistency on the part of the examiner; possible unreliability of the patient or the relatives; and oftentimes lack of cooperation on the part of the family physician. Frequently the observations of the type of patient under discussion are not very reliable and their remembrances may not be accurate. The physician who has previously had them under observation may have noted significant features which would be of great value. Unfortunately these observations are often difficult to obtain. This may be due to failure to keep accurate records or to lack of appreciation of the potentialities of the case and the significance of what are apparently trivial signs and symptoms. In somewhat the same way we may be handicapped by lack of data when called in consultation or asked to operate without having the opportunity of thoroughly studying the case ourselves. Here the fault is primarily our own, being due to a false conception of ethical relations, or fear of offending the attending physician. This is especially so when called to another locality in consultation. This may necessitate working at a disadvantage with unknown consultants and untried facilities; while the time element involved in travel and the lack of accessibility afterwards may prevent a thorough study of the case.

*Read before the American Laryngological, Rhinological and Otolological Society, Inc., Atlantic City, N. J., May 29, 1930.

Brain abscess is met with infrequently in the average practice and its very rarity may contribute to a tendency to overlook its early symptoms. Many times it is a failure to apply indicated diagnostic studies, which results in a missed diagnosis of brain abscess. This may be due to overlooking significant features in the history, failing to obtain all possible data relative to the case, not having the case under constant intelligent observation, or to seeing it only under unfavorable conditions.

Of course all this is obvious, and yet this seems to be a common source of error. Even with the most scrupulous attention to the details of the history there are bound to be many difficulties. The symptoms of intracranial suppuration, such as headache, vague chills, vomiting and malaise, are quite common to suppuration elsewhere. Correct evaluation of the disproportion of symptoms, stressed by Eagleton, requires extremely good judgment. Often definite indications, such as a subnormal temperature, convulsions, evidence of a protective meningitis, or any localizing signs are conspicuous by their absence.

Too frequently, with a case of chronic suppurative otitis media, our investigations will end with a functional test of hearing, and, if the clinical picture demands, we may proceed to do a radical mastoid without sufficient data regarding the labyrinth, spinal fluid and the possibility of intracranial lesion. The possible traumatic effect of the operation on a latent brain abscess should not be lost sight of. If there is a possibility of a subsequent intracranial exploration we should be prepared for it and the interim should not be unduly protracted, despite the frequently false picture of improvement presented after the mastoid operation. The clues which would suggest the advisability of further studies would oftentimes be found if only the history were searched carefully enough.

The following cases were all failures, in which subsequent analysis pointed to significant events in the history which were either unrecognized at the time or not accorded the consideration they merited. Methods of procedure or technic will not be discussed here.

As an extreme example of the handicap imposed by lack of history the following case is cited:

Case No. 1.—Mrs. L. D., age 38. Admitted to hospital on medical service in an unconscious condition. The only data obtained by admitting clerk from the relatives, who immediately departed for their home in an outlying country town, was that she had had severe headache for three weeks and the left arm and leg had been paralyzed for one week. Heart, lungs and abdomen essentially negative. Temperature 99.2 (R.), pulse 94, respirations 20. Involuntary urination and feces. Catheter specimen of urine negative except for slight trace of sugar and acetone. Because of a foul discharge from the left ear otologic consultation was requested. Canal normal. Membrana tympani thickened and slightly reddened, with a round perforation in the superior posterior quadrant, through which was protruding a bit of granulation tissue. No mastoid edema. Right ear normal. There was an apparent paralysis of the tongue and pharynx. Pupils contracted. No nystagmus. Left facial paralysis. Impossible to test for hearing. Neck somewhat rigid. Positive Kernig on the right. No clonus or Babinski. Abdominal reflexes weak. Complete paralysis of left arm and leg. White blood count, 22,000, with 93 per cent polymorphonuclears. Fundus examination (Dr. H. F. Hill) showed definite choked discs. Lumbar puncture not performed.

Operation.—Left mastoid opened, disclosing a necrotic lateral sinus with a thrombus. Culture later showed a growth of streptococcus. The inner wall of the sinus was carefully examined but no erosion could be made out. The dura of the temporosphenoidal lobe appeared normal. Curetting the sinus obtained free bleeding from the torcular but none from the bulb. The patient's condition now made further procedure inadvisable. Death occurred the following day.

When the husband appeared to claim her remains, we were able to get for the first time a history of pain in the left ear four weeks before, followed by violent chills, high fever and headache. Later she developed a left hemiplegia, incoherent speech, involuntary urination and unconsciousness. There were no convulsions.

Necropsy (head only).—Permission obtained with great difficulty. As the body had been taken to the home town, this was done in a little country undertaking room, expecting that the relatives waiting outside the door might interfere at any

time. Necessarily the examination was hurried. This disclosed a thrombosed left lateral sinus and a localized area of meningitis below the right frontal region. On sectioning, a fairly large nonencapsulated abscess was discovered above this area in the posterior part of the frontal lobe. The abscess had not ruptured into the ventricle or the subarachnoid space, death evidently occurring from encephalitis. We were not allowed to take the brain with us for more careful study.

Comment.—This was evidently an acute metastatic abscess of the frontal lobe, resulting from a contralateral otogenic sinus thrombosis. Eagleton* mentions Bleyl's report of a similar case. According to his observations, the route in this case was via the pulmonary circulation, as the cortex was not involved. This would account for absence of convulsions. The apoplectiform onset was quite characteristic. The facial palsy, ipsilateral to the ear lesion, would suggest a contralateral brain abscess or ipsilateral labyrinthine or cerebellar involvement. The hemiplegia pointed to the former. On the other hand, papilledema was more suggestive of cerebellar involvement but was accounted for by the finding of a sinus thrombosis.

Case No. 2.—W. H. T., age 41. No information furnished by referring physician, his family doctor.

History: Discharging left ear since childhood. Deafness. No tinnitus or vertigo. Headaches in past, none recently. Three weeks before had a severe twitching beginning in right hand and ascending to shoulder. Felt numb and was unable to move arm. Lost consciousness momentarily. Similar attack two weeks later. Has felt well otherwise. Right-handed individual. No aphasia, amnesia or mental disturbances.

Examination showed a left chronic suppurative otitis media with a foul discharge. Membrana tympani missing; granulations in attic. Whispered voice heard at three feet. Rinne test 13/17. Weber lateralized to left. No spontaneous nystagmus. No facial weakness. Gait and coordination normal. Reflexes normal. Pulse rate, —70. Temperature normal. X-ray (Dr. J. P. Goodrich) showed infantile sclerosed mastoids. Fundus exami-

*Eagleton: "Intradural Complications of Aural and Nasal Origin." (Archives of Otolaryngology, July, 1926, Vol. 4, p. 76.)

nation (Dr. H. F. Hill) normal. Visual fields not examined, as patient was in a hurry to return to his home, a considerable distance away. A report was sent to his physician, stating the conditions found with the possibility of intracranial involvement, and asking for any data or observations which might have any bearing on the case. No reply was received.

Four days later a telephone message was received stating that he wished to be operated upon the next day. He entered the hospital late that evening. Overlooking the necessity for further study, a radical mastoid was done, under nitrous oxide and oxygen anesthesia. Cholesteatome found in antrum. Dura uncovered over antrum and appeared normal. No leads indicative of intracranial extension found. Primary skin graft performed.

He reacted well and was out of bed on the third day. That evening he complained of headache. Temperature normal. Pulse had ranged between 70 and 90. Next morning he had a convulsion, lasting about five minutes, followed by severe headache. When seen a little later temperature was 102. He seemed clear mentally. No aphasia noted. Neck slightly rigid. Slight Kernig. No clonus. Packing immediately removed from cavity, which appeared to be in good condition. Graft remained in position. Fundi negative. Before anything further could be done patient had another convulsion and died.

Necropsy (head only) disclosed a large flabby walled abscess involving almost the whole left temporosphenoidal lobe. This had ruptured into the ventricle.

Comment.—According to Eagleton,* brain abscesses causing convulsions in adults are always metastatic. The normal appearing dura noted at operation would seem to favor this rather than an adjacent abscess. The lack of a sensory aphasia in a right handed man made the diagnosis of temporosphenoidal lobe abscess less likely, although Eagleton,† quoting Lund in his Survey in the Archives, notes that one-half of his cases of left temporosphenoidal lobe abscess have shown no aphasia. One might also expect some facial weakness on the opposite

*Eagleton: "Brain Abscess." MacMillen, 1922, p. 25.

†Eagleton: "Intradural Complications of Aural and Nasal Origin." (*Archives of Otolaryngology*, August, 1928, Vol. 8, p. 223.)

side. This might have been a metastatic abscess, without much surrounding zone of encephalitis, but near enough to the cortex to account for the convulsions. It was unfortunate that neither visual fields, spinal fluid nor the labyrinth were examined. The lack of abnormalities in the routine examination, the patient's apparent well being, and the absence of data from his physician or confirmatory observations from anyone who had witnessed his epileptiform attacks caused me to neglect the most significant feature of the case. Some time later the referring physician told me that several months before he had treated the patient for what he considered as grippe. He had had slight fever and vague chills, headache and vomiting, possibly the onset of the intracranial invasion. This had not been mentioned previously by the doctor nor admitted by the patient or his wife during what I considered as persistent questioning in taking the history.

Case No. 3.—Mrs. S., age 65. Seen in consultation in hospital. History: No previous ear trouble. Left otitis media of 19 days' duration following influenza. M. T. incised two days after onset. Left facial paralysis noted twelve days before. Urine negative. Blood sugar 140. White blood count, 11,600. Polymorphonuclears, —81 per cent. X-ray: "Large pneumatic double deck mastoid, some obscuring, no breaking down. First degree mastoiditis." Temperature night before, 101; first elevation above 99. Right ear normal. Purulent discharge from left ear, flattening of canal wall and thickening of mastoid periosteum. No spontaneous nystagmus. No hearing test had been done and no tuning forks were available, but voice was apparently heard at one foot. Operation advised. This was done the same morning under nitrous oxide and oxygen anesthesia. Bone was found softened but not extensively broken down. Pus and granulations throughout mastoid, normal dura exposed over antrum. Sinus plate appeared normal. According to hospital records, patient reacted well, and was discharged home 27 days later, apparently convalescent, although facial paralysis persisted. Temperature remained normal, pulse always about 80.

Seen again in consultation, 34 days after leaving hospital and 61 days after operation, having been readmitted. History of being well until two days before, except for muscular

weakness and some vertigo. Has had "fair hearing in ear." Two days before, had nausea and vomiting and had been treated for gastrointestinal upset. Temperature, 101. The next day had increased vertigo, vomiting, headache, and temperature arose to 104 at night. When seen, patient was delirious. Involuntary urination. Neck somewhat rigid, positive Kernig, no clonus, Gordon or Oppenheim. Plantars negative. Middle ear resolved and mastoid about healed. No nystagmus. Unable to test for hearing, due to patient's condition. Examination of eyegrounds requested, but attending physician, who did eye work, was unable to report on fundi, "owing to restlessness of patient not permitting examination." Because of probability of cerebellar involvement, lumbar puncture was not done. Mastoid immediately reopened and dura of middle fossa exposed over whole mastoid. This appeared normal. Sinus exposed and found normal. Cerebellar dura forward of sinus in Troutmann's triangle showed a necrotic area. Pus was evacuated spontaneously through this necrotic dura, with some herniation of brain tissue. Large abscess cavity found in communication with opening in dura and extending inward about 2 cm. Rubber drain inserted. Patient did not react and died that night. No necropsy obtained.

Comment.—Acute mastoiditis with early involvement of facial nerve, later developing cerebellar abscess. Careful tests of cochlea, static labyrinth, fundi and visual fields should have been done before undertaking operation. The weakness and vertigo noted later should have led her physician to consider cerebellar abscess before signs of compression made the diagnosis obvious. There should be no such thing as "stomach upsets" with the possibilities of intracranial abscess. The herniation through the necrotic dura was sufficient evidence of the increased pressure within the cerebellum.

Case No. 4.—J. S., age 59. The writer had been called out of town to perform a radical mastoid on this patient. There was no history of previous ear trouble. Four weeks after being treated for an infected canal the patient had returned complaining of pain, headache and increased discharge from the left ear. Had "loss of memory for names." In examining canal the local otologist passed a probe into a fistulous tract in the superior wall which evidently led into the attic. There

was a large perforation in the posterior inferior quadrant of the drum. X-ray showed sclerosed mastoids. Fundi normal.

Radical mastoid disclosed pus under pressure and cholesteatoma in antrum. Dura found exposed and covered with granulations over antrum. Exposure increased until normal dura uncovered. Usual radical procedure. Advised considering intracranial exploration in 48 hours.

Improved for next two days, after which he began to run a temperature averaging 103, with pulse rate of about 110. White blood count, 14,000. When seen again in consultation, on the fifth day after operation, he was mentally cloudy and had amnesia and sensory aphasia. Pupils equal and reacted normally. Some rigidity of neck. Kernig negative. Operation immediately performed. A large adjacent abscess in the temporosphenoidal lobe was drained through the mastoid approach.

Improved the next day. Mentally clear. Aphasia disappeared. Twenty-four hours later complained of severe headache, went into coma and died. No necropsy obtained, but attending physician reported that he examined operative field after death, finding the drain in place in a large cavity which easily admitted his whole thumb.

Comment.—At the onset, the canal infection distracted attention from the consideration of a chronic otitis media and its possibilities until the fistulous tract to the attic was noted. At this time the amnesia did not inspire the investigative efforts that it should have. Visual fields, labyrinth and spinal fluid had not been examined. As there was only two hours between trains, the time element influenced operation without the thorough study the case should have received. Bearing in mind the amnesia before noted, the condition of the dura might have justified exploring the temporosphenoidal lobe. The subsequent improvement for a few days after mastoid operation, so common in these cases, caused postponement of further interference. The case was not seen again until the fifth day, when conditions were obviously very bad. The trauma incident to the mastoid operation undoubtedly stirred up the abscess so that it was about ready to rupture into the ventricle at the time of the second interference.

It is interesting to note that none of these cases showed either a subnormal temperature or a slow pulse, although possibly a two-hour pulse chart might have shown bradycardia at times.

In a general way, our problem is to try and improve our diagnostic acumen. To do this we may accomplish a good deal by taking a careful, searching history, spending all the time necessary, confirming this by observations of the relatives, and endeavoring to take advantage of the impressions of the family physician or other previous medical attendants. Realizing the difficulties of obtaining this cooperation oftentimes, I have felt that something more than the usual letter must be used. Consequently I am trying the experiment of enclosing a questionnaire with the letter reporting the case. This calls attention to the question of intracranial complication and requests the physician to check any of the enumerated symptoms or signs he may have noted in the case, giving their time of onset. These include the usual symptoms of brain abscess. There is also a place for him to make any additional observations he may see fit. A stamped and addressed envelope is enclosed so there is little excuse to neglect this. This seems to accomplish a threefold purpose. Besides being of help in obtaining valuable data on the individual case, it seems, in my own experience at least, to exert a beneficial influence on the examiner, to keep in mind a little better the possibilities of complications. Its other accomplishment is to make the family physician a little more responsive to the importance of these cases and to what may seem to him quite trivial and unimportant symptoms.

PROFESSIONAL BUILDING.

....., 193...

Patient.....

Dear Doctor.....:

Will you please check any of these symptoms you may have observed on the above-named patient and give duration of each? Your assistance in this matter is of vital importance in establishing a correct diagnosis as there is a question of intracranial complication. Please give this your immediate attention.

.....

Headache.....	Fever.....
Chills.....	Subnormal Temperature.....
General Malaise.....	Slow Pulse.....
Vomiting.....	Projectile Vomiting.....
Change of Disposition.....	Drowsiness.....
Slowed Cerebration.....	Delayed Speech.....
Delirium.....	Convulsions.....
Amnesia.....	Aphasia.....
Anosmia.....	Deafness.....
Vertigo.....	Nystagmus.....
Tremors.....	Ataxic Gait.....
Paralysis, ocular-motor.....	Hemiplegia.....
Paralysis, facial.....	Abnormal Reflexes.....
Duration of focus.....	
(Ear, Sinus, etc.)	

Remarks:

.....

.....

.....

.....

(Signed)

XLV.

A BACTERIOLOGIC AND CYTOLOGIC STUDY OF
THE MAXILLARY ANTRUM IN CHILDREN,
WITH A CLINICAL STUDY OF 83 CASES.*

By BYRON J. ASHLEY, M. D., AND WESLEY V. FRICK, B. S.,†

PORTLAND, ORE.

Due to the high incidence of sinus infection among children on the Pacific Coast, this work was undertaken to determine the relationship of the bacteriologic to the clinical, cytologic and pathologic findings. We are cognizant of the fact that such usage of the word "cytologic" is not entirely correct, but we are continuing its use, by reason of the precedent established by Darling,¹ in 1909, and Sewall,² in 1928. Owing to the fact that the maxillary antrum is the most commonly infected in childhood, and as it is the most accessible, we have confined our studies to that sinus.

Since the beginning of the twentieth century the medical profession has become increasingly aware of the significance of sinus disease. Much work has been done throughout the world in an effort to determine the etiology of such conditions and their clinical significance. One of the most complete of early bacteriologic and clinical investigations on the accessory sinuses of the nose was reported by Lewis and Logan Turner³ in 1905. In their work the pus for examination was obtained with sterile swabs, either from the cavities of the antrum dur-

*Presented by B. J. A. in partial fulfillment of the degree of Master of Medical Sciences, Graduate School, University of Pennsylvania, and W. V. F. toward the degree of Master of Arts, Graduate School, University of Oregon. This work was carried out under the supervision of Harry J. Sears, Ph.D., Professor of Bacteriology and Hygiene, and Ralph A. Fenton, M.D., Clinical Professor of Otolaryngology. The clinical material for the study was obtained from cases treated at the Doernbecher Memorial Hospital for Children of the University of Oregon Medical School. Pathological studies were made by the Pathology Department.

†From the Departments of Bacteriology and Otolaryngology, University of Oregon Medical School, Portland, Oregon, 1930.

INTRODUCTION AND REVIEW OF PREVIOUS WORK.

ing operation, or from the middle meatus of the nose after posturing the patient. Swabs from the nasal chamber were obtained from 42, and direct swabs from 27 of the 57 cavities investigated. The bacteriologic technic used was as follows:

- (a) Films were made from the pus and stained by the Gram method.
- (b) The swab was placed in 1 per cent peptone broth and incubated for 24 to 48 hours at 37 degrees C. The broth usually contained more than one variety of bacteria.
- (c) The material from the broth tubes was diluted and plated out as follows:
 1. Jelly or serum agar.
 2. Blood plates.
- (d) For differential diagnosis the following culture mediums were used: milk, glucose broth, blood serum, blood agar, plain agar, potato and plain broth. Two other mediums, stab jelly and taurocholate, were used, which were not described by the authors.
- (e) Pathogenicity of the various organisms was tested by injecting the material into guinea pigs, rabbits or mice. In certain cases the investigation was supplemented by cultures grown under anerobic conditions. They examined 80 specimens from 57 cavities. The organisms found were as follows:

1. Pneumococcus	42 times	71%
2. Staphylococcus	40 "	70%
3. Streptococcus	43 "	75.4%
4. Diphtheroids	8 "	14%
5. Bacillus proteus	6 "	10%
6. Bacillus mesentericus.....	6 "	10%
7. Bacillus aureus	5 "	8%

Other organisms found less frequently were: *Aspergillus*, *bacillus subtilis*, *bacillus coryza segmentosa*, *bacillus pyocyaneus*, *Friedlander's bacillus*, *bacillus influenzae*, *bacillus zero-sis*, *bacillus hoffmanni*, *bacillus perfringens*, *bacillus coli*, *sarcina*, *leptothrix* and *bacillus buccalis maximus*.

Lewis and Logan Turner⁴ further reported, in 1910, a series of 70 specimens from operations and from washings of the

nasal sinuses through the nose. In addition to the technic described above, these workers made an examination for the tubercle bacillus and for anerobic organisms. The pathogenicity was tested, as above, by animal inoculation. In this series, the bacteria most frequently found were: staphylococcus, streptococcus and pneumococcus. They found anerobic organisms in 19 out of 43 cavities examined. These were represented by both bacilli and cocci.

They conclude that:

1. Sinus suppuration is not caused by any one organism.
2. The pyogenic cocci—i. e., streptococci, staphylococci, pneumococci and micrococcus catarrhalis are the types found most frequently.
3. In bilateral sinus infection the two sinuses may contain different organisms.
4. In recent cases 60 per cent of streptococci found were virulent; while of those found in chronic cases 30 per cent were virulent.

Babcock,⁵ writing in 1918, reported the bacteriologic findings in 100 cases of sinus disease. The material used by the author was obtained by catching the secretion in a sterile tube, as it came from the nose while the sinus was being irrigated. Pure cultures of the following organisms were found:

ACUTE CASES.

Pneumococcus	24
Streptococcus	2
Staphylococcus	10
Bacillus aureus	1
Diphtheroid	1
Micrococcus catarrhalis	2
No growth.....	4

CHRONIC CASES.

Pneumococcus	3
Streptococcus	4
Staphylococcus	15
Bacillus mucosus capsulatus.....	1
Diphtheroids	1
Micrococcus tetragenus	1
No growth	1

In conclusion, Dr. Babcock states that as yet the bacteriologic findings are of some value in chronic cases, but are neither of value in prognosis nor in determining the type of treatment in acute cases.

Dean and Armstrong,⁶ in 1919, were the first to definitely establish the relationship of sinus disease in infants and children to systemic conditions. These authors developed a technic which they believe yielded cultures free from contamination from the nasal mucosa. A cannula was introduced into the antrum beneath the inferior turbinate. A sterile needle was passed through this and sterile water syringed in and out of the sinus. This fluid was examined bacteriologically. Out of 55 apparently normal cases, 44 washings were returned clear, three contained mucus and eight contained pus. Sterile cultures were found in 39 cases, hemolytic streptococcus in one case, hemolytic streptococcus with other bacteria in two cases, and bacteria other than hemolytic streptococcus in 13. In 12 cases showing local evidence of sinusitis, but no systemic lesions, fluid from irrigation was clear in two cases, contained mucus in 8 and pus in 2. Cultures showed hemolytic streptococcus in 3 cases, hemolytic streptococcus and other bacteria in 3, and bacteria other than hemolytic streptococcus in 6 cases. In 12 arthritis cases they found the sinus washings contained mucus in 10 cases and pus in 2. Pure cultures of hemolytic streptococcus were found in 6 cases, hemolytic streptococcus and other bacteria in 6. Out of 98 cases of apparently normal sinuses, except for slightly blurred X-ray plates in 43 cases, the cultural tests showed that bacteria were present in 35 cases or 51 antrums. Out of this number staphylococcus was found in 45 washings, pneumococcus in 13, unidentified gram negative bacilli in 8, diphtheroids in 7, micrococcus catarrhalis in 7, hemolytic streptococcus in 5, streptococcus viridans in 1, and Friedlander's bacillus in 2. Forty-two cases with positive local findings and systemic lesions showed clear washings in 10 cases, mucus in 25, and pus in 7. Sterile cultures were found in 8 cases. Bacteriologic findings showed hemolytic streptococcus in pure culture in 10 washings, hemolytic streptococcus and other bacteria in 9, while bacteria other than hemolytic streptococcus were found in twelve. Twelve of these were from

cases of arthritis, in all of which hemolytic streptococci were found. Improvement was noted after treatment of the sinuses.

Crowe and Thacker-Neville,⁷ in 1919, reported the bacteriologic findings from 70 cases of maxillary sinus infections which came to operation over a period from 1912 to 1918. In this series they found:

SERIES I.

1. Streptococcus	33
(a) hemolytic in.....	10
2. Pneumococcus	12
3. Staphylococcus	16
(a) hemolytic in.....	1
4. Influenza bacillus.....	15
5. Bacillus proteus	1
6. Diphtheria bacillus	2

The same authors also reported a series of 30 cases occurring during February, March and April, 1919, in which the antrums were irrigated by means of a trocar and cannula. The last series occurred during the influenza epidemic. The following results were secured:

1. Streptococcus	14
(a) hemolytic	4
2. Influenza bacillus.....	8
3. Hemolytic staphylococcus.....	4
4. Pneumococcus	3
5. Micrococcus catarrhalis	2
6. Gram negative diphtheroid.....	3
7. Diphtheria bacillus.....	1
8. Bacillus lactis aerogenes.....	1
9. Bacillus proteus vulgaris	2

E. Watson-Williams,⁸ in 1929, also showed the close association between prevailing general infections and sinus disease. He noted a high incidence of pneumococcal infections in the sinuses during an epidemic of pneumonia. This author used the technic employed by his father, P. Watson-Williams,⁹ in 1912, and contemporaries, of introducing a cannula and syringing sterile water into the sinus and withdrawing fluid for microscopic and bacteriologic examination.

The bacteria most frequently found in sinus infections of children as noted by Aland,¹⁰ in 1929, are:

1. Staphylococcus45—80%
2. Streptococcus pyogenes40—76%
3. Pneumococcus30—36%
4. Bacteria occasionally found:
 - a. Influenza bacillus.
 - b. Diphtheroids.
 - c. Friedlander's bacillus.
 - d. Meningococcus.
 - e. Bacillus coli.
 - f. Bacillus pyocyaneus.
 - g. Bacillus pyogenes fetidus.
 - h. Tubercle bacillus.
 - i. Micrococcus catarrhalis.

The clinical or bacteriologic methods were not noted.

Wirth¹¹ reports the results of bacteriologic investigation in 56 cases of nasal accessory sinus disease. The material was obtained by saline washings and at operation. The method of washings was not described nor were the bacteriologic findings from the washings differentiated from those of the mucous membranes obtained at operation. The bacteriologic findings were:

1. Influenza bacillus 15
2. Hemolytic streptococcus 14
3. Friedlander's bacillus 5
4. Pneumococcus—
 - (a) Type 4..... 5
 - (b) Type 1..... 1
5. Staphylococcus aureus 1
6. Diplococcus crassius 2
7. Fusiform bacillus..... 3
8. Anerobic gram negative bacillus..... 1
9. Gram negative rod, like influenza..... 1
10. Gram positive cocci (blood agar only).... 1
11. Nonhemolytic streptococcus..... 1
12. Sterile (mucopurulent exudate)..... 6

In conclusion, Dr. Wirth considers it likely that bacteria infecting sinuses increase in virulence. He also states that bacteriologic investigation of secretions is of prognostic value.

A large series of operative cases was reported by Kistner¹² in 1929. His material was obtained by:

1. Swabs from sinuses opened.
2. Fluid aspirated from cysts found in the sinuses.
3. Culture of tissue removed.

The growths in order of frequency are: Streptococcus, staphylococcus, Friedlander's bacillus, micrococcus catarrhalis and influenza bacillus. There was little difference noted in growth from the swabs and from tissues, except that the swabs showed more mixtures. From the cases without pus, streptococcus was found in 96 per cent. In cases with pus, staphylococcus was always present, but found in pure culture only in cases of empyema. Streptococcus was found in the majority of serous cysts. (The method used in identifying the organism is not described in Kistner's paper.)

Skillern¹³ quotes the following bacteriologic findings, in the order of their frequency, from Hajek and Zarnico. No technic is described.

HAJEK.

1. Influenza bacillus.
2. Pneumococcus.
3. Staphylococcus aureus and albus.
4. Streptococcus pyogenes.
5. Bacillus coli.
6. Pseudodiphtheria bacillus.
7. Bacillus pyocyaneus.
8. Friedlander's bacillus.
9. Meningococcus intracellularis.

ZARNICO.

1. Pneumococcus.
2. Staphylococcus and streptococcus.
3. Influenza bacillus.
4. Pseudodiphtheria bacillus.
5. Friedlander's bacillus.
6. Meningococcus.

7. *Bacillus coli*.
8. *Bacillus pyocyaneus*.
9. *Bacillus pyogenes fetidus*.

Skillern concludes the following:

1. Pathogenic microorganisms are never continually present in normal sinuses.
2. Primary infective bacteria may disappear, allowing secondary infections to continue the disease.
3. Pure cultures of one variety of bacteria are rarely found in chronic cases of sinus suppuration.
4. The commonest organisms found are staphylococcus and streptococcus.
5. Two to five separate and distinct microorganisms usually can be isolated from the same culture.
6. The culture is nearly always contaminated by one or more of the so-called nonpathogenic microorganisms.

It is noteworthy in a review of the literature that a great variety of organisms are reported from sinus infections. No organism is widely accepted as occurring most frequently. Many authors report from two to five organisms from the same sinus washing. We believe, in view of such reports, that either the clinical or bacteriologic technic has been at fault. Dean⁶ states that ordinary antrum puncture and the washing of the contents through the nose is not satisfactory for bacteriologic examination.

CLINICAL TECHNIC.

The wide variation in bacteriologic findings, as reported by previous authors, would indicate bacterial contamination. Any findings in a work of this type, to be of value, should be accompanied by a full description of the technic used. In view of this we have tried to develop a technic which would minimize such possibilities. The material for examination in this work was obtained by means of an antrum puncture beneath the inferior turbinate. During the first part of the work an effort was made to sterilize this region by irrigating the nose with normal saline, followed by swabbing with mercurochrome, hexaresorcinol or metaphen. During that time swabs were taken at the site of puncture and subsequently cultured. In every instance heavy growths of many different organisms were ob-

tained, but no effort was made to identify the species. This would indicate that sinus secretions taken from the nose are unreliable for research investigation. Dean's antrum cannula was used in the beginning, through which a long blunt needle was inserted into the antrum. Five or six cubic centimeters of normal saline were syringed in and out six or eight times and finally transferred to a sterile centrifuge tube for laboratory study. The subsequent washings of the sinus, caught in a black basin, were examined grossly for turbidity or pus.

It occurred to us early in the work that the surface of a large cannula pushed through a heavily infected field might tend to carry more contamination into the sinus than would a smaller needle. In view of this, we substituted a No. 19 spinal puncture needle and omitted the cannula. The syringe was fitted to this by means of a short rubber connection.

The majority of the cases were washed under local anesthesia, using cocain and adrenalin, with the patient sitting in a chair. Children under six years of age, or those older who refused a local anesthetic, were given chloroform. Some cases of adenotonsillectomy were washed at the time of operation, where gas and ether were used. Under the general anesthetic sinus puncture was done with the patient in the prone position, following the technic of Dean and Armstrong.⁶

We feel that our technic has largely eliminated the probability of nasal contamination. We admit that a few organisms may be contaminations from the nasal mucosa, but we believe that such contaminations constitute a negligible percentage of the whole. The following reasons may be given to support this statement:

1. Eighty-seven per cent of all bacteria found were present alone, while in the remaining 13 per cent only two organisms were found in each antrum.
2. The high percentage of negative cultures (67 per cent).
3. The close relationship between negative culture and negative cytology, and positive culture and positive cytology. This will be discussed later in the paper.

BACTERIOLOGIC TECHNIC.

The primary objects of the bacteriologic study undertaken in this work were:

1. To record the frequency in which different organisms are found in the maxillary antrums of children.
2. To make a fairly detailed study of the morphologic and cultural characteristics of the bacteria found.
3. To record the cytologic findings of such sinuses, which would give some indication of infection.

The pathogenicity of the organisms for lower animals was not demonstrated nor were serologic tests carried out, except for typing the pneumococcus found.

The value of the data obtained in such a bacteriologic study as this is naturally dependent upon the reliability of the laboratory procedure used and the care exercised in isolating and identifying the organisms studied. We feel justified, therefore, in describing our bacteriologic methods in some detail.

Washings from the maxillary antrums were received in sterile centrifuge tubes as soon after taking from the patient as possible. In the interim, before reaching the laboratory, they were placed in an incubator at 37 degrees C. On an average such specimens were examined within two hours. The material was first examined grossly and its important characteristics noted. After thorough centrifuging the supernatant fluid was poured off, and the residue was used for the following procedures:

- (a) Microscopic examination of sediment unstained.
- (b) If pus was present, a smear was stained with Wright's stain and a differential count made.
- (c) Smears stained by the Gram and methylene blue methods were examined.
- (d) The material was cultured on the following mediums:
 - (a) Horse blood agar streak plates.
 - (b) Human blood agar streak plates.
 - (c) Plain agar streak plates.
 - (d) Chopped meat medium*—amounts up to one cubic centimeter were inoculated.
- (e) In special cases a glucose infusion agar was used.

*This medium was prepared from finely ground fat free beef heart placed in a culture tube to the depth of one inch. This was covered by two inches of infusion broth and the PH adjusted to 7.

Swabs from operative cases were streaked directly on blood agar and plain agar plates if they were received in fresh condition, otherwise they were placed in glucose infusion broth and incubated over night. Subsequent streaks were made from this material. Membranes from operative cases were treated as follows:

(a) Thorough grinding under aseptic conditions, in a mixture of sterile saline and fine sand.

(b) The finely ground material was pipetted into a sterile tube.

(c) Streaks were made from this material in the manner indicated above, and large amounts were inoculated into the chopped meat medium.

The plates were incubated for 24 hours and examined at the end of this time. Growth was carefully observed, and the characteristic colony formation noted. Notes were made in regard to the hemolysis on blood plates, appearance of the colonies, and any chromogenic characteristics. At the end of 24 hours typical colonies were picked off and the pure culture preserved by streaking on suitable mediums. A Gram stain was made at this time. The plates were then returned to the incubator and examined daily until after 96 hours had elapsed. During this time any changes in the growth were observed. The chopped meat medium was examined, at the end of 72 hours, by means of a Gram stain and streaks on blood agar plates. This medium was especially examined for possible anaerobic or microaerophilic bacteria.

For identification the usual bacteriologic methods were employed. All streptococci were tested for fermentation of the following sugars: Maltose, lactose, saccharose, inulin, glucose, mannite and salicin. All staphylococci were tested for gelatin liquefaction. No species was reported that was not actually isolated and studied in pure culture. The presence of polymorphonuclear and mononuclear leucocytes, red blood cells and epithelial cells was carefully investigated throughout the series. This was done by examination of an unstained smear of the sediment.

BACTERIOLOGIC FINDINGS.

The bacteriologic examination of antrum washings from 83 children resulted in 45 (54 per cent) positive findings. A total of 211 different washings were cultured, of which 72 were positive and yielded 82 separate organisms. Of the above 211 washings, 108 represented only one bacteriologic examination of material from any sinus, 32 washings were from sinuses examined twice, while 13 were from those examined three times. Seventy-two (87 per cent) of the bacteria found in the sinuses were present alone, while ten (13 per cent) were from cavities containing two organisms. This finding, together with the high incidence of negative cultures (67 per cent), gives a good index to the reliability of our technic, both in obtaining the secretions from the patient and in the bacteriologic procedures. We believe that numerous reports in previous work (Lewis and Logan Turner,³ Crowe and Thacker-Neville,⁷ Aland¹⁰ and Wirth¹¹), indicating that several different bacterial species were generally found in infected antrums, should be questioned. As discussed previously in this paper, the nasal passages contain a large and varied bacterial flora, some of which remain after determined efforts to sterilize the mucous membrane, as noted by Bloomfield.¹⁴ The method of catching antral secretions from the ostia and from washing the secretions through the nose therefore is unreliable. The method of Dean and Armstrong, which has been used in a modified form in this work, seems to avoid this difficulty. It is regrettable, also, that many previous papers do not describe the technic used and therefore cannot be considered for comparative purposes.

The relative incidence of the different bacteria found in our series of 211 antral washings is given in Table 1:

TABLE I.

BACTERIAL FREQUENCY CHART.

Bacterial Species.....	Times Found	Percentage
<i>Staphylococcus aureus</i>	23	28.2%
<i>Micrococcus catarrhalis</i>	18	21.1
<i>Staphylococcus albus</i>	10	12.5
<i>Streptococcus hemolyticus</i>	7	8.6
<i>Streptococcus viridans</i>	10	12.5

<i>Streptococcus fecalis</i>	2	2.4
<i>Bacillus influenzae</i>	2	2.4
<i>Bacillus mesentericus</i>	2	2.4
<i>Diplococcus pneumoniae</i>	2	2.4
<i>Bacillus hoffmanni</i>	1	1.3
Unidentified bacteria	3	3.6
<i>Bacillus pyocyaneus</i>	1	1.3
Total	82	100 %

Staphylococcus was by far the most frequently found organism, being present in 40.7 per cent of all positive cultures. *Micrococcus catarrhalis* was found 18 times, or in 21.1 per cent. The streptococci taken collectively also form a large group of organisms, 23.3 per cent of the total positive cultures. This percentage of streptococci is lower than that reported by most authors, but compares favorably with that found by Dean and Armstrong⁴ in a series similar to ours. The highest percentages of streptococci found were reported by Kistner¹² and Crowe and Thacker-Neville.⁷ They found 96 per cent and 47 per cent, respectively, in material secured chiefly from operative cases.

The remaining bacteria found were represented in small numbers, and are of those types which have been previously reported in the literature. This group, also, represents organisms which are normally found on the nasal mucous membranes and may be thought to have gained access to the sinus at an opportune time. We believe that all but a few of the bacteria reported played a definite role in the etiology of the sinus infection from which they were isolated. Proof of this is brought out most satisfactorily by a comparison of positive cultures with the cytologic findings. We have found a decided correlation between the presence of polymorphonuclear and mononuclear leucocytes and the presence of bacteria. This is illustrated in Table 2:

TABLE II.

CORRELATION OF POSITIVE BACTERIAL FINDINGS WITH CYTOLOGY.

Bacterial Species	No. Cultures	Positive cytology	Polys	Monos	Epith.
<i>Staphylococcus aureus</i>	23	18-78%	14	8	12
<i>Staphylococcus albus</i>	10	7-70	7	0	5
<i>Micrococcus catarrhalis</i>	18	18-100	18	7	12

<i>Streptococcus viridans</i>	10	9-90	8	4	2
<i>Streptococcus hemolyticus</i> ..	7	6-95	6	1	6
<i>Streptococcus fecalis</i>	2	2-100	1	1	2
<i>Diplococcus pneumoniae</i>	2	2-100	1	2	1
<i>Bacillus influenzae</i>	1	2-100	2	1	0
<i>Bacillus pyocyaneus</i>	1	1-100	1	0	0
<i>Bacillus hoffmanni</i>	1	1-100	1	0	0
<i>Bacillus mesentericus</i>	2	1-50	1	0	0
Unidentified bacteria.....	3	1-50	1	0	0
Totals	82	70%	59	27	41
Percentages.....	100%	85.3%	72%	33%	50%

It will be seen from Table II that relatively few positive cultures were found in which some evidence of infection was not discovered in the washings. This is particularly true of the larger groups of bacteria found—i. e., the staphylococci, streptococci and micrococcus catarrhalis, which are associated with positive cytology in a high percentage of cases. We can conclude from these findings that the organisms isolated from these antrums play a definite role in the etiology of the condition, either as primary or as secondary invaders. We may also conclude that organisms generally considered to be nonpathogenic, as illustrated by staphylococcus albus, bacillus mesentericus and micrococcus catarrhalis and bacillus hoffmanni, assume a role of considerable importance in sinus infections. All of these organisms were found together with polymorphonuclear and mononuclear leucocytes, which we believe is an indication of their pathogenicity. In a similar work, still unpublished, on adult sinuses, we found several incidences of acute sinusitis associated with such organisms. Our work has led us to believe that bacterial invasion of the sinus is secondary to a lowered immunity, due to a generalized bodily disturbance. Daniels, Armstrong and Hutton¹⁵ and Shurly¹⁶ suggest vitamin A deficiency as a possible factor, while Fenton¹⁷ showed that swimming must be considered. Dean¹⁸ adds the following as possible factors: poor hygiene, improper clothing and ventilation, climatic conditions, metabolic and endocrine disturbances, nephrosis, tonsils and adenoids, nasal blockage and infection.

A further correlation of the bacteriologic to the cytologic findings is given in the following series of tables. We have divided the entire series into four groups:

- (A) Positive cytology with negative cultures.
- (B) Positive cultures with negative cytology.
- (C) Negative cytology with negative cultures.
- (D) Positive cultures with positive cytology.

TABLE III.

POSITIVE CYTOLOGY WITH NEGATIVE BACTERIA.

Cytology Found	No. Times Found	Percentage
Mononuclear leucocytes.....	37	53%
Polymorphonuclear leucocytes.....	22	31
Polys and Monos.....	11	15.6
Epithelial cells.....	26	27

In this group (Table III) we have 70 washings out of 211 (34 per cent), which show positive cytology with negative bacteriology. We feel that such results are indicative of hidden infection in the membrane, and therefore inaccessible to antrum lavage. In one-third of the cases we have positive evidence of an acute process, namely, the presence of polymorphonuclear leucocytes. Mononuclear leucocytes are found in this group in a much larger percentage, being present in over half of the cases. According to Kistner¹² and Sewall and Hunnicutt,¹⁰ such cytologic findings would indicate chronic infection, which would be looked for in the submucosal tissue. These authors, also state that such types of infection are most frequently caused by streptococci. It is unfortunate that we did not secure a larger number of operative cases in order to check our results.

TABLE IV.

POSITIVE CULTURES WITH NEGATIVE CYTOLOGY.

Bacterial species	Present in all Culture mediums	Present in only one culture medium
Staphylococcus albus.....	1	2
Staphylococcus aureus.....	1	4
Streptococcus hemolyticus.....	1	0
Bacillus mesentericus.....	1	0
Streptococcus viridans.....	1	0
Totals.....	5	6

This group (Table IV) represents positive cultures found in washings which gave no evidence of an abnormal condition. Furthermore, six of these organisms were found only in the chopped meat medium, into which a large amount of the original material had been inoculated. We have concluded that the six staphylococci found only in the chopped meat broth medium can be definitely classified as contaminations, picked up either at the time of securing the specimens from the patient or during the laboratory manipulations. On the other hand, the remaining bacteria of this group undoubtedly represent actual infection which may have progressed to suppuration at a later date. None of this group was examined a second time.

NEGATIVE CYTOLOGY AND NEGATIVE CULTURES.

This condition was present in 76 out of the 211 washings, or in 36 per cent.

TABLE V.

POSITIVE CULTURES AND POSITIVE CYTOLOGY.

Cytology Found	No. Times Found	Percentage
Polymorphonuclear leucocytes.....	35	65%
Mononuclear leucocytes.....	2	3
Polys and monos.....	17	31

Fifty-four washings were found to be in this group (Table V), representing 25 per cent of the total number of washings. This group shows a definite correlation between the presence of polymorphonuclear leucocytes and positive bacteria. The percentage of mononuclear leucocytes is very low, and can be contrasted with Group A, in which they occur most frequently. We can conclude, therefore, that the presence of polymorphonuclear leucocytes gives a reliable indication of acute infections.

The importance of epithelial cells in sinus washing is difficult to determine. For this reason we have not included such findings under our positive cytology. There is, however, a definite relation between the presence of such cells in the washings and infections, as indicated by the following table:

TABLE VI.

EPITHELIAL CELLS IN SINUS INFECTIONS.

	No. times Epith. Cells Found	Percentage
Negative cultures, positive cytology.....	26	37%
Neg. cultures—neg. cytology.....	8	10
Pos. cultures—pos. cytology.....	34	64
Pos. cultures—neg. cytology.....	3	27

That epithelial cells are present in washings from normal sinuses has not been substantiated in this work. We have found that in the presence of any infectious process the number of epithelial cells in the sinus washing is greatly increased.

The streptococci isolated in our series were identified primarily from the colony appearance, absence or presence of hemolysis on blood agar—and morphology. As stated before, sugar fermentation tests were made in all cases as a check on other diagnostic methods used. No serologic tests were run.

All staphylococci were tested for fermentation on the different sugars and for gelatin liquefaction. We found that 18 out of 23 strains of staphylococcus aureus liquefied gelatin, while only 2 out of 10 strains of staphylococcus albus produced liquefaction. Eight of 23 cultures of staphylococcus aureus were markedly hemolytic, and four others showed slight hemolysis.

Other bacteria were identified by their cultural and morphologic characteristics, reaction on sugar mediums, motility and growth on special mediums when indicated.

In the work forming the basis for this paper we were interested in determining the presence of anerobic bacteria in infected sinuses. Little mention has been made in previous work concerning them, with the exception of Lewis and Logan Turner,⁴ in 1910, who found anerobes in 19 out of 43 sinuses. We feel that a sinus in which the ostia had been occluded by infection would produce a cavity in which the pressure would be reduced. Under such circumstances one might expect to find anerobic organisms. In our work we used a special chopped meat medium to determine the presence of such organisms but none were found.

We were also interested in determining the difference in bacterial growth on human and horse blood plates, especially bacteria which had been recently isolated from the body. We ran parallel plates of horse and human blood agar throughout the series, but were unable at any time to detect any difference in growth or in hemolytic activity.

CLINICAL DISCUSSION.

This series includes the investigation of 83 cases, from August, 1929, to April, 1930. Thirty-eight cases, or 93 antrums, had negative bacteriologic cultures of the washings, and 45 cases, or the washings of 72 antrums out of 124, had positive cultures. The majority of these patients were seen in the outpatient clinics, and sinus irrigations were performed only when considered necessary either for treatment or diagnosis. Consequently there are only six cases with no nasal complaint or pronounced local findings. However, the X-ray was positive in four of these.

The percentage of positive cultures, as shown in the table, indicates the prevalence of upper respiratory infections during the school months when the children are in steam heated buildings. December and January show a variation because of snow and low clinic attendance most of these cases are from the hospital.

TABLE VII.

SEASONAL INCIDENCE OF POSITIVE CULTURES.

	Cases Washed	Negative Cultures	Positive Cultures	Percentage Pos. Cultures
August	4	3	1	25%
September	10	5	5	50
October	11	5	6	54
November	16	5	11	68
December	6	3	3	50
January	6	5	1	16
February	15	6	9	60
March	14	6	8	57
April	1	0	1	100
	83 cases	38 cases	45 cases	54%

NEGATIVE BACTERIOLOGIC FINDINGS.

The cases are divided into two main groups. First, those in which the culture of the sinus washing showed no bacteria

at any time (Table IX). Second, those in which the culture of the sinus washing showed bacteria in one or both sides (Table X). A further division was made in the latter by grouping those antrums in which the bacteriologic examinations of the washings were negative. These will be referred to as from the positive bacterial group.

A fair comparison of these two negative bacteriologic groups cannot be made, because many of the antrums in the subdivision of the positive group have on occasion shown bacteria in the washings. However, it is interesting to note the difference in percentage of results of cytologic examinations and the secretions found on washing. (Table VIII.)

TABLE VIII.

NEGATIVE BACTERIOLOGIC CULTURES.

38 Cases or 93 Antrums (Negative Bacteriologic Group)	26 Cases or 48 Antrums (Positive Bacteriologic Group)
Positive cytology 39 antrums.....42%	22 antrums46%
11 antrums, positive 1 side only)	
Polymorphonuclears alone, 10 antrums10.7%	7 antrums14%
Mononuclear leucocytes alone, 24 antrums.....24.5%	3 antrums..... 6%
Polymorphonuclear and Mononuclear leucocytes together, 4 antrums 4%	3 antrums 6%
Epithelial cells alone, 10 antrums.10.7%	10 antrums20%
Positive X-ray, 29 cases.....87%	19 cases90%
X-ray not taken, 5 cases..... 5%	4 cases 8%
2 cases, yellow pus in washings.	4 antrums, yellow pus in washings.
5 cases, mucopus in washings.	18 antrums, mucopus in washings.
18 cases, turbid washings.	18 antrums, turbid washings
	7 antrums, clear washings
	1 antrum, blood in washing.

From the first or negative bacteriologic group with positive cytology, irrigation and X-ray, 18 cases, or 28 antrums, have a history of continual low grade nasal disturbance with frequent acute exacerbations. Bacteria may or may not be present in the membrane, as was noted in case No. 93, where

staphylococcus aureus was found in the membrane of one side only; and in No. 96, where the membrane of each antrum was positive for bacteria. Mononuclear leucocytes were found on cytologic examination in 18 antrums, being alone in 8 and with epithelial cells in 8, which would indicate mononuclear leucocytes are found associated with low grade processes.

Positive washing, cytology, X-rays and negative bacteriologic findings were found in 10 patients, or 16 antrums, bacteria having been present at one time in 9 of the antrums, or 7 patients. Cytologic examination showed polymorphonuclear leucocytes in 12 antrums, mononuclear leucocytes in 5. Epithelial cells were found only in conjunction with polymorphonuclear leucocytes. This would show the relationship of polymorphonuclear leucocytes to recent or active conditions. It is questionable whether bacteria are present in the membrane at this time. The high percentage of polymorphonuclear leucocytes present might lead to the belief that submucosal penetration of bacteria had occurred.

Negative cytology, negative cultures and positive irrigation were noted in 10 cases, or 12 antrums. However, the washings of six of these sinuses contained epithelial cells on microscopic examination. This is an interesting group. Both membranes from case No. 83 cultured hemolytic staphylococcus aureus, which was also found in fluid from a cyst.

Staphylococcus aureus was present in both membranes of case No. 95. In case No. 125 cystic fluid obtained at time of irrigation was negative for bacteria, but at operation the fluid from a cyst on the same side, obtained with a sterile syringe and needle, cultured streptococcus viridans and staphylococcus aureus. Case No. 106 gives a history of sinus infection with several positive washings. The above mentioned are cases of chorea. A bilateral Caldwell-Luc operation was done on the first two. The X-ray was positive, and lipiodol injection showed thickened membranes except in the right side of patient No. 125, where it was questionable. Virulent infection may be present in a sinus, even though the washing be clear or show only a slight turbidity and only epithelial cells be found on cytologic examination. The X-ray finding does not have to be marked as noted in the right antrum of case No. 125.

Turning to those in the positive bacteriologic series,^{*} with negative cytology and bacteriology, positive irrigation and positive X-ray, 10 cases, or 17 antrums, were found. Two had chronic suppurative otitis media; one case, no local findings, and one, a recent cold. The culture from the opposite side was positive only in the broth medium in four cases. The low grade process in these cases accounts for the negative cytology or presence of epithelial cells alone.

Negative washings and cultures with positive cytology and X-ray were noted in 9 cases, or 15 antrums. Cytologic examination showed ten sinuses with mononuclear leucocytes and five with polymorphonuclear cells in the washings. These children had continual nasal disturbance and positive local findings. Nos. 31 and 47 gave a history of previous purulent maxillary sinusitis. In a chorea case, No. 93, where a bilateral Caldwell-Luc operation was done, *staphylococcus aureus* was cultured from each membrane. Membranes obtained by radical operation from case No. 54, with only epithelial cells in the washings, gave *staphylococcus albus*. The membranes were thickened and hyperplastic, with a marked infiltration of polymorphonuclear leucocytes beneath the epithelium. At subsequent washings it is probable that leucocytes might have been found. Clear washings may be associated with positive cytology and positive bacteria in the sinus membrane. The preponderance of mononuclear leucocytes in these chronic processes is noteworthy.

Negative X-ray findings, negative cultures with positive irrigation and cytology were found in three cases, or four antrums, from the positive bacteriologic series. Two of these children, No. 9, aged 4, and No. 128, aged 7, had Still's disease. Although we have secured repeated positive cytologic findings in these patients, we have been unable to demonstrate any notable changes by flat radiographs. It is possible that stereoscopic X-ray plates might have been helpful in these instances.

CASES GIVING POSITIVE CULTURES.

There were 45 children, or 74 antrums, with positive bacteriologic cultures from sinus washings.

Six of these cases gave a positive history but no local findings. Five of these gave a history of recent colds. Four patients had polymorphonuclear leucocytes on cytologic examination and five had epithelial cells. X-ray findings were negative in three cases. The bacteria found were streptococcus hemolyticus once alone, micrococcus catarrhalis six, three times alone, staphylococcus aureus three, alone two times, staphylococcus albus 3 times, and bacillus influenzae one. The history of recent colds, the presence of bacteria and the predominance of polymorphonuclear leucocytes and epithelial cells would indicate the membrane of the sinus had not returned to normal, even though the nasal examination was negative. Micrococcus catarrhalis was most commonly found.

Thirty children, or 46 antrums, had positive cultures, irrigation, cytology and X-ray plates. Thick yellow pus characterized the washings in five of the cases, or seven antrums, three of which showed polymorphonuclear leucocytes alone, three both polymorphonuclear and epithelial cells, and polymorphonuclear, mononuclear and epithelial cells in one. Bacteria found in pure culture were streptococcus viridans one, bacillus mesentericus one, staphylococcus aureus two, staphylococcus albus one, and streptococcus hemolyticus one. None of these children had systemic lesions. As noted above, polymorphonuclear leucocytes were present in all antrums. Mucopus was found in the washings of nine cases, or seventeen antrums. The cytologic findings in four were polymorphonuclear leucocytes alone, polymorphonuclear and mononuclear leucocytes in two, polymorphonuclear and epithelial cells in nine, and polymorphonuclear, mononuclear and epithelial cells in one, polymorphonuclear leucocytes being found in all but one sinus. Pure cultures of bacteria were streptococcus viridans four, streptococcus hemolyticus three, micrococcus catarrhalis two, bacillus pyocyaneus one, gram positive branched bacillus unidentified one, staphylococcus albus one, and streptococcus viridans with staphylococcus albus one. Four cases had chronic suppurative otitis media, one arthritis and one acute nephritis and subacute mastoiditis. All of these cases had streptococci in the sinus washings. The predominance of polymorphonuclear leucocytes with an active process is noted. The finding of streptococci in associated systemic conditions is suggestive

that the sinus may be the original focus and is of importance with reference to treatment. *Micrococcus catarrhalis* was frequently associated with recent colds. Turbid washings were present in seventeen antrums and ten contained red cells. Polymorphonuclear leucocytes were found in twenty-two sinuses and alone in six. Epithelial cells in sixteen and alone in one. Mononuclear leucocytes in ten and alone in one. The bacteria found were *micrococcus catarrhalis* six times and with *staphylococcus aureus* or *staphylococcus albus* three times, *staphylococcus aureus* six times, *streptococcus hemolyticus* two, *streptococcus viridans* two and with *staphylococcus aureus* one, *diplococcus pneumoniae* (type III) once alone and with *staphylococcus albus* one, *bacillus influenzae* one and with *staphylococcus aureus* once. History of a recent cold was obtained in four of the cases with *micrococcus catarrhalis*, streptococci being found in those cases with systemic lesions or the more virulent upper respiratory infections. *Staphylococcus aureus* in pure culture or associated with other bacteria was found in a number of sinuses but it seems to be related to a local process rather than systemic lesions.

Negative irrigation with positive cultures were present in nine cases, or fourteen antrums. The radiographic study was negative in seven of the sinuses. No X-ray picture was taken in three cases. Polymorphonuclear leucocytes were found in eight antrums, four of which were with mononuclear leucocytes; four had epithelial cells alone. History of a recent cold was obtained from five children. Bacteria found were *micrococcus catarrhalis* six times alone and with *staphylococcus albus* two, *staphylococcus aureus* two, *streptococci* once, *bacillus hoffmanni* one, *staphylococcus aureus* two, and *streptococcus fecalis* (*enterococcus*) once. None of these cases had systemic lesions. *Micrococcus catarrhalis* was found only in those cases with history of recent colds. Patient No. 54 had a bilateral Caldwell-Luc operation. The left antrum, which had *bacillus hoffmanni* in the culture, is included in this group. The membrane showed only a moderate edema with cystic dilatation of submucous glands. There was a slight infiltration of lymphocytes, plasma cells and occasional eosinophiles. The culture was negative. The findings on the right side have been noted previously.

Negative radiographs with positive cultures were present in twelve cases, or fifteen antrums. Cytologic examination showed polymorphonuclear leucocytes in eleven, epithelial cells in ten and mononuclear leucocytes in four antrums. The washings were clear in seven and turbid in five. The negative X-ray, negative irrigation and high percentage of polymorphonuclear leucocytes, with the presence of bacteria would indicate recent acute processes, no marked change in the membrane having occurred. Four children gave definite history of recent colds. The bacteria found were staphylococcus albus six, streptococcus fecalis (enterococcus) one, diplococcus pneumoniae (type III) one, bacillus pyocyaneus one and bacillus influenzae one.

Negative cytology or merely epithelial cells accompanied positive irrigation and local findings in seven cases. The X-ray picture was positive in four and not taken in two.

CONCLUSIONS.

1. The normal maxillary sinus does not contain bacteria.
2. Nonhemolytic staphylococci are most commonly found in the maxillary sinus and are related to local processes rather than systemic lesions.
3. Various streptococci, not always hemolytic, are associated with systemic lesions.
4. Bacteria may be present in the membranes though the washings are bacteriologically negative.
5. Bacteria may remain in a sinus for some time following an upper respiratory infection, with little or no clinical findings and negative radiographs.
6. Micrococcus catarrhalis is commonly present in the maxillary sinus following acute colds.
7. Bacteria, in most cases, play a role secondary to other factors in the etiology of sinusitis.
8. A pathologic condition may be present in the antrum with only epithelial cells in the washings.
9. Turbid washings are as significant in diagnosis as those containing pus or mucopus.

10. Polymorphonuclear leucocytes in sinus washings indicate a present or recent active process.

11. A predominance of mononuclear leucocytes indicates a chronic low grade process.

BIBLIOGRAPHY.

1. Darling, J. M.: *Edinburgh Medical Journal*. Series 3, 3:542, 1909. Cytologic Examination of the Discharge in Cases of Suppuration of the Maxillary Sinus as a Guide to Treatment.
2. Sewall, E. C.: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:642 (June), 1928. Diagnosis and Treatment of Chronic Maxillary Sinus Infection.
3. Lewis, C. J., and Turner, A. Logan: *Edinburgh Medical Journal*, 18:393, 1905. Suppuration in the Accessory Sinuses of the Nose. A Bacteriological and Clinical Research.
4. Lewis, C. J., and Turner, A. Logan: *Edinburgh Medical Journal*, Series 3, 4:293, 1910. A Further Study of the Bacteriology of the Suppuration in the Accessory Sinuses of the Nose.
5. Babcock, J. W.: *Laryngoscope*, 28:527 (July), 1918. Bacteriological and Clinical Aspects of Infections of the Accessory Sinuses of the Nose.
6. Dean, L. W., and Armstrong, M.: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 28:452 (June), 1919. Sinus Disease in Infants and Young Children, Including a Bacteriological Study.
7. Crowe, S. J., and Thacker-Neville, W. S.: *Bull. Johns Hopkins Hosp.*, 30:322 (Nov.), 1919. Influenza Bacillus in Paranasal Sinus Infection.
8. Watson-Williams, E.: *British Medical Journal*, 1:720 (April), 1929. Variations in Incident of Pneumococcal Infections in the Nasal Accessory Sinuses.
9. Watson-Williams, P.: *Journal of Laryng., Rhin. and Otol.*, 27:142, 1912. The Diagnostic Value of the Suction Syringe in Maxillary Antral Sinusitis.
10. Aland, A. H.: *California and Western Medicine*, 27:74 (July), 1927. Sinusitis in Children.
11. Wirth, E.: *Zeitschrift für Laryngologie*, 16:453, 1928. Bakteriologische Befunde bei Nebenhöhlenentzündungen.
12. Kistner, F. B.: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 38:795 (Sept.), 1929. Chronic Nonpurulent Sinusitis and Its Clinical Significance.
13. Skillern, Ross H.: *Accessory Sinuses of the Nose*, 1923, Page 28.
14. Bloomfield, A. L.: *Bull. Johns Hopkins Hosp.*, 34:65 (Feb.), 1923. The Effect of Antiseptics on Bacterial Flora of the Upper Air Passages.
15. Daniels, Amy, Armstrong, L. M., and Hutton, Mary K.: *J. A. M. A.*, 81:828, 1923. Nasal Sinusitis Produced by Diets Deficient in Fat Soluble A Vitamin.

16. Shurly, Burt R.: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 37:251 (March), 1928. Vitamins and Dietetics in Relation to Otolaryngology.

17. Fenton, Ralph A.: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, 32:526 (June), 1923. Sinusitis from Swimming.

18. Dean, L. W.: *J. A. M. A.*, 93:838 (Sept.), 1929. Nasal Sinus Infection in Children.

19. Sewall, E. C., and Hunnicutt, L.: *Arch. Otolaryng.*, 10:1 (July), 1929. Cytologic Examination of the Antrum.

TABLE XA. POSITIVE BACTERIOLOGICAL GROUP.

Case number.	2	5	9	13	14	18	23	24	25	28	30	36	39	40	42	43	44	49	50	54	55	56
Month.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.	Aug.
Day.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.
Age.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.	13.
Sex.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.
Side.	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL	HL
Chief complaint.																						
Colds.																						
Nasal obstruction.																						
Nasal discharge.																						
Pain.																						
Suppuration.																						
Serous.																						
Headache.																						
Vertigo.																						
Arthritis.																						
Hepatitis.																						
Chronic otitis media.																						
Fever.																						
Diabetes.																						
Duration over three months.																						
Clinical findings.																						
Artery.																						
Positive irrigation.																						
Nasal mucosa.																						
Nasal polyp.																						
Positive cytology.																						
Polymerbuccular leukocytes.																						
Mononuclear leukocytes.																						
Epithelial cells.																						
Congested mucous membranes.																						
Nasal obstruction.																						
Nasal secretion.																						
Pain.																						
Suppuration.																						
Positive allergic tests.																						
Tonsils and adenoids in.																						
Progress.																						
Number of cultures.																						
Number of irrigations.																						
right side																						
left side																						
subacute																						
adenoid present																						
positive or present																						
Negative or not present																						
nothing done																						
turbid																						
blood																						

TABLE XB. POSITIVE BACTERIOLOGICAL GROUP--Continued

129 128 124 114 113 114 129

TABLE XB. POSITIVE BACTERIOLOGICAL GROUP—Continued.

[illegible]

XLVI.

A SIMPLE METHOD OF KEEPING SINUS X-RAY RECORDS.

By JOHN A. PRATT, M. D.,

MINNEAPOLIS.

Owing to the city regulations for keeping X-ray films, it was found necessary to devise some simple method of registration as to the size of the frontal and the degree of infection of the different sinuses.

The simple rubber stamp (made by the Hoff Rubber Stamp Co., 224 S. 4th St., Minneapolis) seems to have solved the problem. The cut that has to be made first can be used for printing new records and the rubber stamp to register on the old records. The size of the stamp is $2\frac{1}{4}$ by $1\frac{1}{2}$ inches.

The size and shape of the sinuses with their variations in contour can be drawn, using the space below the chin for the sphenoid.

When the X-ray is questionable, the outlines are drawn and a question mark is placed in the space.

Diagonal lines drawn one way designate the sinus cloudy.

Diagonal lines drawn in both directions designate the sinus dark.

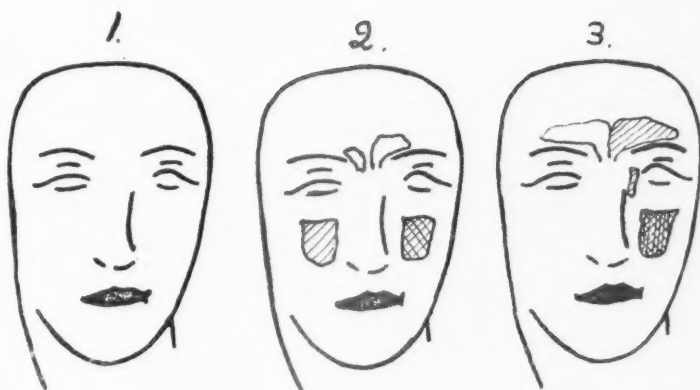
Diagonal and perpendicular lines designate the sinus black.

Cut No. I shows the stamp with no registration.

Cut No. II shows a small right frontal and a medium sized left frontal, but both are negative. The right antrum is cloudy, as shown by the single diagonal lines, and the left antrum is dark, as shown by the double diagonal lines.

Cut No. III shows large frontal sinuses with the left one cloudy. The ethmoidal sinus on the left side is cloudy. The left antrum is black, as shown by the diagonal and perpendicular lines.

1753 MEDICAL ARTS BUILDING.



XLVII.

LARYNGEAL DIPHTHERIA.*

By V. K. HART, M. D.,

STATESVILLE, N. C.

The O'Dwyer intubation set has been a life saving means of no little importance. Many cases go on to uneventful recovery. A number of others have a stormy convalescence; indeed oftentimes serious complications occur. These latter cases are more numerous than is usually appreciated by the profession. Ultimate recovery is made surer and chances of complications are diminished by certain measures commonly overlooked.

These measures are executed routinely: 1. Pagenstecher linen is used and is not cut but is pasted to the cheek and tied round the ear. This allows for quick withdrawal in case of obstruction by secretions, food or membrane coughed from subglottic area. It also diminishes trauma to the larynx in removing the tube. Children soon become accustomed to the linen in the mouth. 2. Just enough atropin is given to check excessive salivation. 3. A properly fitted tube is used. Too large a tube is apt to produce subsequent laryngeal stenosis. Too small a tube is easily dislodged by coughing. 4. Cuffs are put on both arms to prevent child's pulling out the tube. 5. Sufficient antitoxin is administered. Never is less than 20,000 units given. 6. Small amounts of liquids are given at frequent intervals. 7. Digitalis is used. Recent work of the University of Michigan Pharmacology Department shows this to be one of the best drugs available as prophylaxis against the toxic effects of diphtheria on the heart muscle. 8. Proper hospitalization is resorted to when possible; at least, an experienced nurse is kept in attendance. 9. A tracheotomy set is always at hand when intubation is done and is left in the room afterwards.

*Read before the Eye, Ear, Nose and Throat Section of the State Medical Society at Pinehurst, N. C., May 1, 1928.

Having successfully intubated the patient, when should a test extubation be done? As early as seventy-two hours later; not later than ninety-six hours. This because the tube, though effective in its relief of stridor, is bound to act more or less as a mechanical irritant. Long continued wearing of a tube is apt, being superimposed on an inflammatory condition, to precipitate or maintain extension of the inflammation to the arytenoid cartilages with a resultant edema preventing extubation and possibly going on to a true laryngeal stenosis. In the great majority of cases those patients who are going to get along without tubes can be successfully extubated in seventy-two hours. The majority of cases in which the tube had to be replaced have gone to tracheotomy. Occasionally a case is seen where the tube is worn five to seven days, or even longer, and later successfully removed. If the tube must be used longer than four days, these patients are to be viewed with apprehension.

An average case without complications is first cited:

Case 1.—Female child, age two and one-half years. First seen December 4, 1926. Pharyngeal and laryngeal diphtheria. Definite stridor. Thirty thousand units antitoxin given intramuscularly. Labored breathing so marked No. 2 O'Dwyer tube put in place. Immediate relief. Extubated December 8, 1926. Uneventful convalescence.

A case is next cited illustrating the other extreme and exemplifying the necessity for always being ready to do an instant tracheotomy because of the possibility of subglottic involvement below the tube or because of dislodging a piece of membrane and pushing same below the tube while doing an intubation:

Case 2.—Male, aged three and one-half years. First seen at home October 18, 1927. Marked stridor and cyanosis with all accessory muscles of respiration in play (history showed child had been sick some forty-eight hours before family doctor was called). Immediate intubation with No. 3 O'Dwyer tube gave no relief. Tube taken out and replaced several times with no change at all in breathing. Emergency tracheotomy done. Moved to hospital for post-tracheotomic care. Given 20,000 units antitoxin. Uneventful convalescence and successfully decannulated seven days after admission.

On the other hand, subglottic involvement may come on after intubation, and despite all treatment, as shown by the following case:

Case 3.—Male, age two years, first seen at home August 13, 1927. Pharyngeal and laryngeal diphtheria. Stridor marked and respiration much embarrassed. Intubated with No. 2 O'Dwyer tube and moved to hospital. Immediate relief. Twenty thousand units antitoxin given. Child apparently going on to uneventful convalescence. Thirty hours later an emergency call found the patient with marked stridor and cyanosis. Tube hastily withdrawn (showing value of leaving linen attached). Found patulous. Cleansed and replaced. No relief. Emergency tracheotomy done at once. Successful decannulation one week later and uneventful convalescence.

As previously suggested, a child sometimes cannot be extubated. The sequence of events is reflected in this case:

Case 4.—Male, age three years, first seen at home November 2, 1927. Laryngeal diphtheria obvious. Immediately intubated with a No. 3 tube with complete relief. Family doctor had previously given 30,000 units antitoxin. Unsuccessful extubation on third, fourth and twelfth days. The first two times the tube was successfully replaced with relief, but the third time the stridor and cyanosis became so marked, even after replacing the tube, that it was necessary to do a very rapid tracheotomy. (Would it not have been better to have done it much earlier, removing all mechanical irritation from the larynx and putting same at rest?) Child was moved to hospital for post-tracheotomic care. One week later breathing exercises were started, believing it loosens the musculature of the larynx, stimulates the circulation and promotes absorption. Even after changing from a No. 2 to No. 1 cannula, the child was able to go only an hour or two with the same corked, and then not with complete relief. Exercises were repeated daily, however, and the child sent home after the mother had been instructed rigidly how to daily change the tracheotomy tube, care for same and carry out breathing exercises. A week later, fearing a postdiphtheritic stenosis, a direct laryngoscopy was done with a small Jackson laryngoscope. The motility of the cords was good, there was no granulation tissue or adhesions and no fixation of arytenoids. There was slight residual swelling of right arytenoid. Following this the child slept quietly on three successive nights with the outer cannula corked and decannulation was carried out without further trouble.

Still another case is cited to show the value of early tracheotomy when the child cannot be extubated in five or six days. Tracheotomy was done by necessity almost a week earlier in the course of the disease than in Case 4. Convalescence was shortened at least ten days thereby, because decannulation was begun at the end of one week. (Not until about the seventeenth day after tracheotomy in the preceding case.) Short history follows:

Case 5.—Male, aged three years, brought to hospital November 18, 1927, with unquestioned laryngeal diphtheria. Marked stridor and cya-

nosis. Intubated at once with No. 3 O'Dwyer tube. Complete relief. Twenty thousand units antitoxin given (an undetermined amount had previously been given by family doctor.) Unsuccessful extubation on third day. Marked stridor compelled replacement of the tube. Extubated again on the sixth day. Given syrup of ipecac as expectorant to loosen any residual membrane. Temporary relief only, though given to point of vomiting. Tube was replaced with difficulty because of swelling of arytenoids. (This illustrates a most important point previously mentioned, viz., the mechanical irritation of the tube in exciting or maintaining a perichondritis or chondritis, or both.) Marked stridor and cyanosis continued with tube replaced and an emergency tracheotomy was done. Uneventful convalescence. Successfully decannulated one week later.

All the above cases were unquestioned diphtheria. In such the Klebs-Loeffler bacillus can be demonstrated in cultures from areas of involvement in pharynx or larynx, from membrane on the intubation tube when extubated and from the tracheal secretions when tracheotomy is done. This latter work was carried out with interest for obvious reasons.

There is a type of streptococcic laryngitis which very closely simulates diphtheria, even to the production of a pseudomembrane. A personal case was recently reported.³ Still another case is briefly here recapitulated to show the difficulties of diagnosis:

Case 6.—Called on October 18, 1926, to see baby, age nine months, because of difficult respiration. The breathing was more "stuffy" than obstructed. There was no definite stridor, no cyanosis. The nose gave evidence of an upper respiratory infection and there was a large mass of adenoids in nasopharynx. No membrane was in the nasopharynx or pharynx. Temperature 99 3/5 (axillary). An adenoidectomy was recommended if trouble continued.

That evening a hurry call found the child in collapse. A No. 1 O'Dwyer tube was hastily put in place with no relief. The child stopped breathing with tube in situ. A very rapid tracheotomy was done and artificial respiration started. (This case again shows the importance of always having a tracheotomy set ready with intubation set.) The child rallied, was moved to hospital, No. 1 Jackson tracheotomy tube placed in trachea and usual post-tracheotomic care instituted. Uneventful convalescence and child decannulated one week later.

A study of the tracheal secretions did not show diphtheria organisms. Staphylococci and streptococci can always be secured on culture of secretions of upper respiratory tract. However, this was interpreted as an acute streptococcic laryngitis due to extension from an acute coryza.

SUMMARY.

1. Intubation should be done first. Often nothing else will be necessary. 2. Proper care after intubation is very important. It has the same relation to intubation as post-tracheotomic care to tracheotomy. 3. Never should an intubation be done without a tracheotomy set a hand. It should be left in the room with the intubated patient. 4. A test extubation should be done on the third or fourth day. If necessary, replace and do another test extubation on the sixth day. The return of stridor and cyanosis at this time is a clean cut indication for tracheotomy, even if relief is obtained by replacement of the tube. Long continued wearing of the tube excites or maintains inflammatory processes. Early tracheotomy in these cases diminishes the possibility of postdiphtheritic stenosis of the larynx. 5. Subglottic involvement of much extent is always an indication for tracheotomy. 6. Routine post-tracheotomic care, as outlined by Dr. Jackson, must be followed. 7. A rest of the larynx for one week seems to be very necessary in the average case requiring tracheotomy. 8. It is believed that laryngeal exercises should be started as early as one week after tracheotomy, even if the outer cannula can only be partially corked at the start. It is best to change to a size smaller tube first. The time of corking is increased each day as the patient's comfort allows it. Such exercises probably hasten absorption and diminish adhesive processes. 9. Routine digitalization of all patients with laryngeal diphtheria is valuable prophylaxis against myocardial failure.

BIBLIOGRAPHY.

1. Baum, H. L.: Tracheotomy and Intubation. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY*, 26:472-478, 1927.
2. Edmunds, C. W., and Cooper, R. G.: Action of Cardiac Stimulants in Circulatory Failure Due to Diphtheria. *Journal A. M. A.*, 85: 1798-1801, 1925.
3. Hart, V. K.: Streptococcic Laryngitis. Report of a Case With a Very Rare Complication. *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY*, 36:781-785, 1927.

XLVIII.

PRESIDENT'S ADDRESS.*

BY LEWIS A. COFFIN, M. D.,

NEW YORK.

We are assembled in response to the call of our Council for the fifty-third annual meeting of the American Laryngological Association.

I am mindful of the high honor done me by my election to the presidency of this Association and take this opportunity to thank you, its members, for the vote of regard and confidence which places me in this enviable position.

My first duty, the official announcement of the death of our much esteemed and beloved president of last year, Dr. Charles Richardson, fills me with sorrow and sadness. I am sure that each and every member feels that, in the removal of Dr. Richardson from our midst he has met with a great personal loss. Dr. Delavan, as our historian, will, I am sure, give us a detailed account of Dr. Richardson's life and accomplishments.

Our honored and much respected Secretary of the Treasury of the United States, at a recent birthday dinner, said: "Age comes on so gradually and unnoticed that one can be unconscious of any material change. But seventy-five years of life leads one into a reflective and contemplative frame of mind and makes one, I think, more appreciative of old associations and friendships, as well as more keenly perceptive of the changes affecting present day conditions and habits of life."

Try, as I have, to get away from the reminiscent state of mind, it has been impossible of accomplishment, and in that frame of mind I have during the past year had many pleasurable hours. Hours spent in the contemplation of the men and their work which has placed our specialty on the high plane

*Presented before the Fifty-third Annual Meeting of the American Laryngological Association, Swampscott, Mass., May 22, 23, 24, 1930.

which it occupies today; in contemplation of how at first apparently independent truths and facts may be woven together to form the great fabric known as acquired knowledge and progress. Tonsils have been the subject of study and discussion as to removal and methods of so doing since 10 A. D., whereas the much allied growth, adenoids, were not discovered and described by Dr. Wilhelm Mayer until 1868 A. D., and then the universal recognition and removal of that growth came about quite slowly, largely on account of the lack of standardization of method of removal. Great impetus was given, in this country at least, by the work of Dr. Hooper, who in 1888 presented a paper showing with what frequency the growths occurred and presenting a forceps which made easy the operation, with or without anesthesia.

Strange as it may seem, the man who was one of the first to describe this growth and his method of removal in New York was the most vigorous in disagreement with Dr. Hooper as to the frequency of their occurrence, saying that he thought the frequency with which Dr. Hooper had found the condition must be due to some peculiarity of the climate of Boston. It was Dr. Delavan who said at the same meeting that he was surprised that a man of Dr. Beverley Robinson's standing should so express himself in the face of such overwhelming evidence as presented by Dr. Hooper, Dr. Hooper having reported something over one hundred cases and putting in evidence nearly as many bottles containing the masses removed. This paper was read in the New York Academy of Medicine. I am sure every laryngologist who heard the paper sent to Codman & Shutford, instrument makers of Boston, the following morning for a pair of Hooper's forceps, and the war on adenoids was on, and the multiplicity of adenoid forceps and curettes was begun. The combined operation for the removal of adenoids and tonsils was soon in vogue.

I have presented these facts because of the bearing of tonsils and adenoids on other diseases with which we have to deal. During my last years of doing active hospital clinical work the two subjects which were of the greatest interest and concern to me were "Sinusitis in Children" and "Atrophic Rhinitis and Ozena."

Before discussing the interdependence of these diseases, I wish to call attention to certain historical facts and established truths.

First.—As stated above, the combined operation for the removal of tonsils and adenoids was not in vogue until 1889.

Second.—The first pandemic appearance of la grippe occurred in 1889, occurring in this country in the last quarter of that year and the first quarter of the next. Accompanying and following this disease there were many cases of sinusitis which forced attention of laryngologists upon the pneumatic cavities of the head as had never before been known.

Third.—In a paper by L. W. Dean on "Sinusitis in Children," in which he had had the collaboration of pediatricians and pathologists, he has stated that they found 80 per cent of the chronic cases of sinusitis recovered from simply improving nasal drainage by the removal of tonsils and adenoids. Granting this, how much greater a percentage of acute cases would recover from the same treatment, and if the drainage be prophylactically established how greater a percentage still would be, so to speak, prophylactically cured.

When one considers that in 1889, when this infectious grip was raging, comparatively few children had thus been prophylactically protected, one can see what an extensive and fertile field was ready in the noses and sinuses of the children of the land.

Atrophic rhinitis and ozena had been the subject of much speculation for many years, but during the latter part of the nineteenth and the early part of the twentieth century it was so persistent and so disturbing to the afflicted and his family as to be the *hôte noir* of the laryngologist and a nerve wrecking force to the suffering.

All who have been in our special field of work for over twenty years will recall the many theories advanced as to the cause and cure of this most trying and disgusting disease. Many, and among them some of the best minds and most authoritative writers, looked upon the condition as an independent disease and offered many pages of literature on the pathology, specific producing organisms, etc., to account for the condition. On the other hand, many of our best thinkers

have from early years of the specialty considered it as a complicating sequela of a diseased sinus or sinuses.

A most convincing paper in support of this contention was presented, in 1913, by our fellow member, Dr. Francis Emerson, before the Rhinological, Laryngological and Otological Society.

If you have followed me, you will no longer wonder that we have had such a harvest of this disease in the period to which attention has been called, and you will as well understand why now, when most children needing it have had tonsils and adenoids removed, atrophic rhinitis and ozena have practically been wiped from the map of diseases.

For the same reason we see less of chronic sinus disease and therefore less of radical sinus work done. If this be so, we must add a large credit to the tonsil and adenoid operation. Many, both of our own and other specialists, have been shocked at the frequency with which the operation for the removal of tonsils and adenoids has been done, and we have heard such critical phrases as the slaughter or massacre of the tonsil. We have all, very probably, shared in this feeling at times. However, I think we must blame not the thousands of operations, but the disease. We will all hail the day and the man or organization called for by Dr. Delavan in his paper of last year that will show us the cause and the method of preventing the condition demanding these operations.

To my mind, there is no doubt that, some time, diet or improper feeding of our young will be found to be a contributing factor in the cause of these hypertrophies which become so fertile for infection and so physically detrimental to nasal drainage. I can remember, some twenty years ago, in a discussion of the relationship of gastrointestinal disease to disease of the upper air passage, I said to Dr. Einhorn that if he would show me the throats of his patients I would tell him their diets.

A very interesting and illuminating paper on this subject was read at the Eastern Section of the Laryngological, Rhinological and Otological Society last winter by Dr. Jarvis of this Association.

I heartily agree with Dr. Delavan that this Association could do no more worth-while piece of work than to promote a study of these conditions. To this end there should be appointed a promoting or steering committee consisting of laryngologists, pediatricians, biochemists and pathologists, to direct the study. This much may be said: Our problems multiply, our field widens. The mountains of the unknown invitingly beckon to him who would wrest from them crumbs of knowledge. My belief and abiding faith is that there will ever be men to accept the invitation and thus shall knowledge grow.

Gentlemen, I declare the fifty-third meeting of the American Laryngological Association open for such business as may properly come before it.

DINNER TO DOCTOR DELAVAN.

A dinner was given to Doctor David Bryson Delavan Thursday evening, May 1st, at the New York Academy of Medicine, under the auspices of the Laryngology section, in recognition of his conspicuous services to Laryngology.

Doctor Delavan, a native New Yorker, graduated from Yale in 1872 and from the College of Physicians and Surgeons in 1875, and served his internship at the Charity Hospital. In 1877 he became Chief of Clinic and Lecturer in the treatment of throat diseases in the College of Physicians and Surgeons. During a long and fruitful career Doctor Delavan has held many of the major positions in Laryngology in and around New York City.

For many years Doctor Delavan has been actively interested in the New York Academy of Medicine, having served as Corresponding Secretary and as a member of the Council. The section of Laryngology was organized in 1886 through his efforts.

The use of radium in the treatment of nasopharyngeal fibroma was first suggested by Doctor Delavan in 1915. He also introduced the successful treatment of diphtheria carriers by means of disinfection of the throat with Dakin-Dunham solution of Dichloramin-T.

Doctor Delavan has been elected to many societies. He has been twice President of the American Laryngological Association, is a Fellow of the Royal Society of Medicine, and a member of the British Laryngological Society.

Many letters of congratulation were received from colleagues all over the world, who spoke of Dr. Delavan as one who had an exceptional influence upon laryngology.

The speakers brought out that the guest of honor is interested in many lay organizations, being the President of Grenfell Association of America and the First Vice-President of the American Scenic and Historic Preservation Society.

Abstracts of Current Articles.

Treatment of Cancer of the Larynx (Traitement du Cancer du Larynx).

G. Canuyt (Strasbourg), *Arch. Int. Lar.*, 8:1153, Dec., 1929.

Professor Canuyt states that surgical treatment is his preference for cancer of the larynx; that total laryngectomy should be done less frequently and, with earlier diagnosis, laryngofissure done more often. Laryngofissure should be the first step of laryngectomy in any case. In partial laryngectomy the thyroid cartilage should not be resected unless it has become involved. He prefers a cigar shaped roll of gauze for hemostasis after laryngofissure. This is left in place six or seven days, and prevents food and saliva passing into the larynx. The "cigar" is replaced, and finally removed about the fifteenth day, when the external wound is allowed to close.

Deep X-ray therapy, and particularly the radium collar—properly screened and at some distance from the neck—are valuable in postoperative treatment. He has had no trouble with necrosis of cartilage.

F.

Functions of the Peripheral Labyrinth (Teoria del Funcionamiento del Laberinto Periferico).

R. Lorente de N6 (Madrid), *Rev. Espan. y. Am. de Lar., OI and Rin.*, 20:435, Nov., 1929.

Concluding this series of studies of the present knowledge of the anatomy and physiology of the vestibular nerve, Lorente de N6 states that only the following facts are indisputably proven: 1. That the semicircular canals produce nystagmus; 2. That the semicircular canals produce reflexes to accelerate movement in one direction; 3. That the semicircular canals are concerned in the production of turning reactions; 4. That the semicircular canals are concerned in the production of tonic ocular reflexes.

The first fact depends on endolymphatic currents, the latter three upon displacements of the membranous canals within the bony canals, in his opinion. Regarding the otolith system, he regards the macula as organs which react slowly to changes in position of the head in space. He rejects the theories of

Quix, Magnus and de Kleijn as incapable of experimental proof. His own theories, whose limitations he carefully points out, include the following: Each crista is surrounded by a zone of rather solid attachment to the bony canal, while the crista itself and the remaining membranous canal are very loosely contained within the bone; hence dislocation of the membranous canal wall within the bone is readily affected, while the crista may move up and down but not laterally. Stimulation of a macula is impossible unless deformation of the corresponding otolith membrane can be produced (by gravity or other force). Such deformation he likens to the change in shape of a drop of liquid lying on a surface, without change in area, when the surface is tilted about. De N6 points out the necessity for further study on mammals before these things can be proved. F.

Radiograph of Cancer of the Larynx (Una Radiografia de Cancer de la Laringe).

L. Suñé y Medán (Barcelona), *Rev. Espan. y Am. de Lar., Ot. and Rin.*, 21:25, Jan., 1930.

Lateral views of the normal and cancerous larynx are given, and very accurate notions of the lateral and downward extent of the growth are obtainable. F.

Vascular Relations Between the Subarachnoid Space and the Healthy and Inflamed Nasal Mucosa (Rapporti Vascolari fra gli spazi Subaracnoidei e la Mucosa sana ed infiammata).

A. Bronzini (Pisa).

By injection of india ink into the subarachnoid space of dogs and rabbits by suboccipital puncture, the author found not only intense coloration of the olfactory zone but also of the ethmoid mucosa. Slight reticular coloring was found in the inferior and median walls of the antrums, and rather marked injection in the roof and lateral walls of the pharynx. This coloration became most marked five to six hours after injection. Ink was not observed in the deep cervical glands until three or four days after injection.

Acute inflammations, produced by iodine, acids, hot water, and especially by abrin (jequirity) solution, blocked the dissemination of the ink entirely on the inflamed side. In three dogs one month after very severe inflammation has been provoked and allowed to subside, the ink diffusion proceeded normally;

in other words, lymphatic function was reestablished. These facts were demonstrated histologically as well as in the gross specimens. F.

The Roffo Test for Cancer (La Reaction de Roffo).

G. Portmann (Bordeaux), *Rev. de Lar., Otol., Rhin.*, 51:12, Jan., 1930.

Professor Roffo of Buenos Aires, after a long series of experiments on growing normal and neoplastic tissue, found that neutral red is set free from disintegrating tumor tissue much more rapidly than from normal tissue similarly stained. Applying this to study of the blood serum, he found that five drops of 1:1000 neutral red (Grübler) in distilled water, added to 1 cc. of serum, produce a yellowish color in normal individuals, but in cancerous persons the color rapidly becomes an intense red. Prof. Portmann reports results of numerous clinicians, which show positive results agreeing with the clinical diagnosis in an average of 65 per cent in one series of 814 cases. Sarcoma runs only 50 per cent accurate. In another series of over 4,000 nonmalignant cases, only 5 per cent of positives were found. Out of 108 benign tumors, seven positives were found. After radical operations in another series in which biopsy was used to prove the diagnosis, the test was found to have changed to negative in some, positive or feebly so in others, according to the completeness of operation and freedom from metastases. F.

Jugular Thrombosis in Children (Les Thrombo-phlebités des Sinus Chez les Enfants).

Moulouquet and Doniol (Paris), *O., R., L. Internat.*, 13:455, Oct., 1929.

One of a series of important papers on otitic septicemia presented at the Latin Otolaryngologic Society meeting last fall in Madrid, this article emphasizes the difficulties of diagnosis and gives a personal mortality of 59 per cent. Metastatic abscess is often the first convincing sign of venous involvement. Fever may drop to normal. Severe spontaneous temporal pain may cause complaint. Lung complications are less frequent than are suppurative arthritis and subcutaneous abscesses. The sinus may rupture spontaneously, with severe hemorrhage. Nasopharyngeal and glandular involvement further complicate the clinical picture, and the prognosis is far more grave than in adults. F.

Biopsy in Cancer of the Larynx (Le Biopsie dans le Cancer du Larynx).

G. Canuyt (Strasbourg).

Professor Canuyt agrees with the elder school of Anglo-American laryngologists that biopsy may be done very readily by the indirect method. He suggests, however, that in cases where the growth is low, laryngofissure may occasionally be done to secure the specimen. He does not agree that biopsy will cause increase of the growth, and counsels a repetition if negative histology is found in the first specimens. Several fragments of the growth should be secured at the first sitting, if possible. Canuyt hesitates to advise radical operation unless he has histologic proof of malignancy. F.

Vestibular Phenomena of Patients Suffering from Morbus Menière.

S. H. Mygind (Copenhagen), Acta Otol., 13:303, 1929.

Basing his conclusions on the recent exhaustive thesis of his pupil, Dida Dederding, on 135 cases of Menière's disease, eliminating all cases with syphilis, mumps, encephalitis, brain tumor and multiple sclerosis, Mygind states that spontaneous nystagmus is always found during such attacks, although it rarely is found between attacks. Point tests were found valueless. Giddiness varies from a vague sense of unsteadiness to the classical apoplectiform seizure. Experienced patients usually recognize prodromal warnings—headache, tinnitus and pressure. Periodicity of attacks is noteworthy.

Since the vestibular tests closely resemble those found in labyrinthitis from otitis media, Mygind considers Menière's disease to be a peripheral, labyrinthine affection rather than a neural or central nuclear lesion—probably an increase of fluid content in the affected labyrinth. Only after repeated attacks of such pressure and stasis do permanent alterations of structure and losses of function occur. F.

Vestibular Problems (Problèmes Vestibulaires).

F. H. Quix (Utrecht), O., R., L. Internat., 13:346, Aug., 1929.

To start discussion before the Latin O. R. L. Society, Quix raises the following questions: 1. The exact origin of the different forms of nystagmus, especially rotatory and vertical; 2. Whether the semicircular canals actually maintain equilibrium; 3. Determination of adequate stimulation for a semi-

circular canal, and of the relation of endolymph currents to varying speeds of head rotation; 4. Accurate explanation of cold and heat reactions: are they due to endolymph currents or are they vasomotor or thermic phenomena? 5. Redetermination of galvanic reactions; 6. Reexamination of the otolith mechanism in man without regard to results of animal experiments. Accurate otolith tests must be devised; 7. Clinical examination of the vestibular organ should include all its elements instead of merely the horizontal canals; 8. More knowledge of the central nuclei and course of the vestibular nerve. Quix requested the appointment of a committee to examine these matters and report at the next Congress. F.

Combined Pharyngo-Laryngeal Paralyzes from Influenza (Laringoplegie Associate di Natura Influenzale).

G. Bettin (*Venice*), *Riv. Oto.-Neuro.-Oft.*, 6:363, July, 1929.

One case involved the ninth, tenth and eleventh nerves of one side; the other included the motor fifth, seventh, ninth, tenth, eleventh and twelfth. Lesions ranged from difficulty in swallowing and deviation of the tongue to paresis of one vocal cord, weakness of one sterno-cleido-mastoid and paresthesia of taste. Bettin considers this syndrome purely toxic peripheral neuritis. Both were cured by salicylates within three weeks.

F.

Professor E. J. Moure of Bordeaux is to be congratulated on the completion of the fiftieth year of publication of his *Revue de Laryngologie*. In the January 15th issue he publishes a very interesting review of the advance of the specialty during his years as editor and teacher.

Professor Citelli, president of the Italian O. R. L. Society, makes the suggestion that not only Italian, but Spanish, shall be added to the three official languages of the next International Congress. Another effort to replace Anglo-American and Germanic prestige by that of the Latin group, this will not popularize the Madrid Congress with English-speaking otolaryngologists.

Society Proceedings.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Monthly Meeting of Monday, February 3, 1930.

THE PRESIDENT, DR. SAMUEL SALINGER, IN THE CHAIR.

DR. ROBERT WEST, professor of speech pathology, University of Wisconsin, by invitation, addressed the Society on

"Neurosis as a Cause of Speech Disorder."

(ABSTRACT.)

Although one cannot say that neurosis is responsible for more speech disorders than all other causes, yet it appears to be blamable for more disturbance of speech than any other single group of causative conditions. The chief reason for the prominence of neurosis as a pathogenic factor in the function of speech is to be found in the emotional coloration of neurosis.

The neurotic is unable to (or at least does not) exert the normal cerebral inhibition upon the centers where emotional movements are subtended, viz., the thalamus and striate bodies. These emotional movements are not engaged in generally by all of the muscles of the body, but are more or less concentrated in the muscles of respiration, mastication, and deglutition, and in the superficial facial muscles. The only muscles of the groups mentioned above that do not play a direct part in speech production are a few facial muscles around the eyes and across the forehead. The use of these groups of muscles for the enacting of their intended emotional roles does not always preclude their playing their learned speech roles. High emotional excitement of any color robs us of the power of skillful and delicate articulation, and certain emotional states block speech entirely. The emotional states most disastrous to good speech are those whose inherited pattern involve muscle movements opposite in direction or greater

in intensity than the movements necessary for speech. Many a case of stammering begins with what seems to be a sobbing mechanism. Other emotional patterns, such as those of fear and anxiety, do not so much involve actual movements conflicting with those necessary for speech, as they pre-empt the muscle groups for other uses by setting them in a state of hypertonicity. The normal functioning of speech requires many movements of extremely light touch. Only the explosives can be made with intense muscle contraction. Other sounds require delicate poise between antagonistic muscles. Any neuroses, therefore, characterized by fear or anxiety tend to interfere with speech by disturbing this poise.

We studied, by means of the X-ray, cases of aphonia for which we could find no possible contributory cause. The neurosis seemed the only explanation of the loss of voice. We found that we were unable to analyze the X-ray pictures until we had developed a series of pictures of subjects whose voice training gave them the ability to hold the laryngeal adjustments in various degrees and combinations of tension.

In certain cases, at least, of hysterical aphonia the neurosis shows itself in a hypertension in the direction of phonation, but beyond the point of efficient voice production. It is this principle of overtension that is a frequent neurotic cause of speech disorder. It is quite possible that, if we talked with our hands, disorders of speech would be less frequent.

DISCUSSION.

DR. ELMER L. KENYON said that Dr. West's method of approach to the etiology of speech disorders was relatively new and not familiar to him. In using the term "functional aphonia," meaning the production of voice by action of the speech mechanism, in which the normal loud quality is totally replaced by a quality closely resembling a whisper, one should bear in mind that the mechanism of speech production acts normally to produce each of these qualities. He had studied closely the action of the peripheral speech mechanism in hysterical aphonia, and had felt convinced that its action was definitely that of the normal whispered voice. If this be true, the nervous disturbance is one that temporarily and functionally

destroys the possibility of producing action of the speech mechanism for the loud quality of voice but does not interfere with normal innervation for the whispered voice. He would feel that such a disturbance probably had its seat in the cortex, and that it was produced by a perfectly normal innervation and muscular action of the speech mechanism for the whispered voice. That such a voice could be imitated by artificially producing wrong muscle action seemed to him possible; but would this method of study throw light on the fundamental neurotic disturbance? At this moment it seemed to him that wrongness of muscular action in stammering, in aphonia and in nasality could be more probably explained on grounds quite different from those suggested by the speaker. But it might be that certain muscular disturbances of various disorders of speech could be explained, at least in part, through neurotic tendencies suggested by Dr. West, and his point of view should be carefully studied.

DR. MEYER SOLOMON (by invitation) said many different terms are used in describing neurosis; here it had been used as a bodily disturbance due to a psychologic cause. In respect to speech disturbance the mind can affect the body in two ways; one, by volition; second, through emotion. By volition it can affect the musculature of the extremities, of the trunk and those parts of the body under volitional control. By emotion it can affect any portion of the body, even the chemistry of the blood, through fear, anger, and so on. One may have speech disorders due directly to volitional control, and others due to emotional disturbance. The first condition is typical of ordinary hysteria. In this condition things occur due to the idea of the individual that he or she is unable to do certain things. If convinced that one is unable to do a certain thing, no effort will be made to do it, and every effort will be made to do everything except that thing. The hysterical person cannot do anything with the body that a normal person cannot do. A malingerer may purposely, consciously, imitate loss of power of some sort. The malingerer knows he can do the things he claims he cannot do, and the hysterical person really believes that he cannot. In hysteria all that can happen to the speech mechanism is that the patient either makes no effort to use the proper muscles or makes the wrong effort.

In the emotional speech disturbances, such as stuttering, there can be any sort of upset in the peripheral speech machinery. It is not the same as hysteria. It is not a purely voluntary state. In stuttering one of the two main things that happens with the peripheral speech machinery is that the individual in his haste produces an attitude in which he is unable to produce any sound at all; or he is able to produce the first sound but is unable to advance to the second sound.

As the speaker saw the problem, the speech machinery can be upset in stuttering and allied emotional disturbances, or in hysteria which is due to autosuggestion. In the latter, no effort is made or the wrong effort is made. Among the wrong efforts is the hypertension that Dr. West had brought out. The idea of utilizing the roentgen-ray in this work was new to him, but no difference what the findings are in the speech machinery one must relate it back to the general psychologic state of the individual.

DR. AUSTIN A. HAYDEN called attention to the fact that the last speaker had at one time stuttered very severely, and the fact that he had completely overcome it was a great achievement and a splendid tribute to the men who work in speech disorders. The general otolaryngologists he thought should be very proud of having among their number men like Dr. Kenyon, who devote their entire time to the remedial treatment of these distressing disorders. The work is very tedious, and often it is only a labor of love rather than remuneration. In his opinion, all the general men need to know of speech disorders is the name, address and office hours of a man who does such work. All patients with speech defects should be referred to these specialists, instead of being operated upon first to cure the defect.

Dr. Hayden was much impressed with the presentation of Dr. West, but thought his points would have been carried much better had he plotted the X-ray findings against the larynx of the normal individual.

DR. ROBERT WEST, Madison, Wis. (closing), referring to the use of the phrase "hysterical aphonia," said that he was not entirely satisfied with the term "hysterical" in this con-

nection and that he used it not because it is accurate but because it is the one used in literature to describe the condition in which aphonia appears without definite structural or physiologic cause. He defined the condition as a neurosis and said that the more he works with patients with speech defects the more he is inclined to think of a neurosis not as something that is caused by an unfortunate incident in the patient's life, but as a condition resulting from a whole complex of situations that affect the neurology and psychology. One of his students, for example, is at present working on an analysis of the blood and urine of the stuttering individuals, and has found a very high glucose content of the blood and a high carbon dioxid content of the urine.

He agreed that many of these patients get well by psychoanalysis, if one could find the disturbing complexes, such, for example, as one that develops because a boy has a marked enuresis over a period of years. The embarrassment that comes from this might cause a speech defect, but he believed if this occurred it was due to something more than the embarrassment. He is trying to find out what the connecting link is—whether it comes through the central nervous system, the blood or what.

As to why some patients can make a consonant sound but cannot go on to the vowel sound, he believed the consonant sound can be made because it is an explosive and the vowel sound is not. The explosive sounds can be made much more easily than the half position necessary for the vowel sound. The neurotic individual seems to have difficulty in leaving a contracted position and going at once to a muscle position in which there is normal muscle balance which allows for phonation. There would be fewer disorders of speech if we talked with our hands, because the hands are not connected with the emotional centers which control the mechanism of speech.

He believed one could say that hysterical speech difficulty may be outside of cerebral direction but not outside of brain direction; certainly not outside of central direction; and quite possibly is influenced by many factors of muscle metabolism.

DR. WILLIAM A. SMILEY read a thesis entitled

"Foreign Bodies in the Nose."

(ABSTRACT.)

Most cases of foreign body in the nose occur in children. Beans, matches, cotton, corks, buttons, tin toys, pencil erasers, being among the most frequently used objects, are readily accessible objects, appropriate for insertion and consequently frequently seen by the physician. Usually the child is frightened by having put some article into the nose and tells the parents, or if too young to talk calls their attention to it by crying, pricking at the nose or rubbing the nose. A large percentage of foreign bodies are removed by the parents immediately after insertion. When they are unable to remove them they usually call for aid at once. However, there are a few cases in which the parents never suspect the presence of a foreign body in the nose, but consult a physician because of some symptom resulting from the irritation of the particular object.

The most frequent symptom is unilateral nasal discharge. The discharge is usually mucopurulent but often serous. At times there is history of bloody expectoration. Nasal obstruction on the affected side is usually present. General symptoms are commonly absent, and when present are due most frequently to the swallowing of pus or blood, which drips backward into the throat. Loss of appetite and loss of weight may be present. Secondary anemia was marked in one of the essayist's cases.

A careful examination is necessary in every case where there is unilateral nasal discharge. The nose should be carefully cocaineized, and if the desired shrinkage of the mucosa does not result adrenalin should be used. The discharge should be removed carefully, and one can usually see an area where there is some bleeding. This should be carefully palpated with a probe if a foreign body cannot be seen. An object which has been in the nose for any length of time will give the sensation of bare bone. Even cotton will become incrustated with a calcareous deposit and yield this sensation.

Except in refractory children, general anesthesia is rarely necessary for the removal. Each object will present its own problem in removal and the method must be decided on in each individual case.

Careful clinical observation is of the greatest value in arriving at the correct diagnosis and intelligent treatment.

DR. LAWRENCE J. LAWSON read a thesis entitled

"Some Diagnostic Considerations When Vertigo Is a Symptom."

(ABSTRACT.)

To properly interpret clinical findings it is often necessary to combine practical theoretical considerations with recognized anatomic findings.

Case 1.—Mr. B. T. B. was admitted to the Evanston Hospital January 25, 1929. The chief complaint was dizziness. One month previous to admission the patient suddenly developed severe pain on the right side of the head, principally back of the right ear. No ear infection was present. Following the initial attacks of pain, there were several attacks of vertigo. There was at no time a loss of consciousness. He was able to reach a chair or bed with little difficulty. His attacks were characterized by blurred vision and extreme vertigo. There was no tendency to fall to a particular side. There was no nausea or vomiting, and no recurrence of the headache. Four days before admission he had a very severe attack of vertigo while at his desk, he felt quite weak, and his associates stated that he was very pale. There was a daily recurrence of these attacks until admission to the hospital. He had been well until the present attacks. The family history was negative for anything related to the problem presented. A neurologic examination was made by Dr. Bassoe the day after admission. The pupils were equal and regular. The optic discs were normal. The deep and superficial reflexes were normal. The Wassermann and Bárány tests were advised, complete rest and no tobacco. The Wassermann test was negative. The external auditory canals were unobstructed, and the drum membranes were intact and gave no evidences of infection. The vestibular tests were given the second day after admission, by caloric stimulation with the patient at rest in bed. Water at 10° centi-

grade was used. Irrigation of the right ear with 30 cc. gave a horizontal rotatory nystagmus to the right of normal amplitude, after 45 seconds, lasting 60 seconds. Irrigation of the left ear with the same amount gave a horizontal rotatory nystagmus to the left of increased amplitude, after 30 seconds, lasting 90 seconds. This was interpreted to mean a noticeable increase in the irritability of the right labyrinth. The patient continued to improve in his freedom from vertigo, had no attacks while under observation, and was sent home after five days to plan an extended rest in Florida. Two days later he was seized with a violent vertigo at night and died ten minutes after readmission to the hospital. At necropsy there was revealed both recent and organized thrombosis of the basilar and right vertebral arteries.

Case 2.—Miss P. K. was admitted to the Evanston Hospital July 9, 1929. This patient had recovered from a bilateral otitis media associated with scarlet fever, only to be precipitated into another bilateral attack by measles. Upon admission to the hospital two weeks after the onset of the second attack, the temperature was 105° F., the leucocyte count was 16,500, of which 78 per cent were polymorphonuclear cells. There was a right acute suppurative mastoiditis, a marked right cervical adenitis, and a subsiding left otorrhea. Roentgenograms revealed necrosis of the right mastoid cells but no changes in the left. The right mastoid was opened on the day of admission, the lateral sinus exposed, but found unchanged, and the dura not exposed. Cultures revealed gram positive cocci in pairs and chains. On the fourth postoperative day the cervical adenitis appeared to be undergoing suppuration, and an incision was made under gas anesthesia, which yielded 20 cc. of pus. On the eighth day there was a distinct phlegmonous state in the right cervical region, and under gas anesthesia three deep incisions were made but no pus was found. The cervical infection decidedly retarded healing of the mastoid wound. On the twelfth day pus came from the deep incisions, and by the fourteenth day there was a striking local improvement, but the patient looked toxic. The patient had complained some of hearing foreign sounds. Hearing in the left ear was somewhat diminished. There was a slight horizontal rotatory nystagmus on looking to the extreme left. Sitting up

or sudden turning of the head produced vertigo. The left drum membrane was intact with no bulging, and there were no acute symptoms referred to the left ear. The patellar reflexes were brisk, there was a bilateral ankle clonus, slightly more pronounced on the left at times, a questionable bilateral Babinski and no Kernig. On the eighteenth day, with local symptoms rapidly clearing, with the temperature, pulse and respiratory rate near normal, the patient looked ill, and symptoms of intracranial pressure became manifest. The spinal fluid revealed 800 cells per cubic millimeter, chiefly polymorphonuclears, and not under noticeably increased pressure. The white blood count was 35,000, of which 76 per cent were polymorphonuclears. Disturbance of coordination, while suggesting cerebellar involvement, did not definitely lateralize the site of lesion. If the predominance of polymorphonuclear cells over lymphocytes in the spinal fluid is taken as an index to poor operative prognosis, this contraindication was present. In the absence of further localizing symptoms, and because of the poor condition of the patient, no intracranial exploratory work was done. The patient expired on the twentieth postoperative day. Necropsy revealed normal right sided postoperative findings. There was no meningitis. On opening the left temporal bone (the symptomless side) an intact drum membrane was revealed, and softening of a few cells high in the petrous portion with an erosion at the internal auditory meatus. There was an abscess stalk from this point of contact to the adjacent cerebellum, the left lobe being filled with pus. There was a localized meningitis and a beginning left sigmoid sinus thrombosis.

Comment: The patient with the thrombosed basilar and right vertebral arteries had transient extreme vertigo with an experimental nystagmus indicating only a slightly increased irritability of the right side. This discordance and the absence of acute ear pathology point to a central lesion. The unsteadiness and tendency to fall during the attacks, without falling definitely to the right or left, are indicative of cerebellar involvement. The experimental horizontal rotatory nystagmus and the conjugate eye movements with equal pupils exclude lesion of the oculomotor brain. An infratentorial lesion was indicated by the extreme vertigo, pallor and faintness.

The patient with the cerebellar abscess had a slight tinnitus, increasing deafness and slight nystagmus on looking to the extreme left, which would have merited more attention had not the presence of so much pathology on the opposite side and the contraindications to vestibular testing masked the situation. The left vestibular and cochlear nerves were probably blocked by the adjacent necrosis, but the left cerebellar abscess would account for the slight continued left nystagmus. These pathologic developments would emphasize further the urgency of repeated roentgenologic examination bilaterally of ear infections running an irregular course.

CASSELBERRY PRIZE FUND.

The sum of \$500.00 having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, said sum is now available, in part or as a whole for a prize award, decoration or the expense for original investigation and research in the domains mentioned above. Theses or reports of work must be in the hands of the Secretary, Dr. George M. Coates, 1721 Pine Street, Philadelphia, before February first of any given year.

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